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TRAUMATIC TRACHEAL RUPTURE

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TRAUMATIC tracheal rupture may result following sharply localized blunt trauma to the upper anterior chest and neck. It is important that this type of injury be recognized early since immediate repair, especially of long lacerations or rupture-separation of the trachea, will bring about prompt restoration of the airway with minimal risk of loss of voice or subsequent tracheal stenosis. Smaller lacerations of the trachea will heal promptly if an immediate tracheostomy is done to prevent passage of air through the tear when the patient coughs or strains against a closed glottis. The late sequelae of untreated traumatic tracheal rupture are tracheal and subglottic stenosis which may necessitate permanent tracheostomy. The technical problems involved in late restoration of an adequate airway in patients having post-traumatic stenosis are formidable but usually can be overcome so that permanent tracheostomy rarely should be accepted as an end result. Hood and Sloan[9] in 1959, in their comprehensive review of the world literature, found only 18 reports of traumatic tracheal rupture. The paucity of literature on this subject prompts the authors to report their experiences with 13 patients having traumatic tracheal rupture; 9 cases involved the cervical portion and 4 the intrathoracic trachea. Not included in this report are 2 patients who had knife wounds of the cervical trachea and one patient having rupture of the trachea from explosion of anesthetic gases.

MODE OF INJURY

There has been considerable speculation as to just what conditions must be present in order to cause traumatic tracheal rupture. Nach and Rothman[13] in

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a collected series of subcutaneous ruptures of the trachea describe instances in which rupture occurred by the head suddenly being thrown back by severe coughing in an attempt to expel a foreign body, as well as by other motions which tend to quickly overstretch the trachea. It has been postulated[16] that the rupture can be caused by a sharp blow to the chest at the time when the glottis is closed so that the trapped air within the tracheobronchial tree is brought under considerable increased pressure causing the trachea to rupture. Rupture of the trachea has been reported as being due to overinflation of the balloon cuff of an intratracheal tube.[2, 10] Explosion of anesthetic gases during anesthesia may have caused tracheal rupture in one patient who was treated by the authors and was not included in the present series. Dubost and associates[4] reported a similar accident in which the patient was successfully treated by immediate suture. Rupture-separation of the cervical portion of the trachea is usually due to a direct force tending to avulse the larynx and cricoid cartilage from the trachea. Intrathoracic rupture of the trachea seems to result from a sharply localized force applied to a relatively elastic chest wall anteriorly, compressing the trachea against the rigid bodies of the vertebrae posteriorly so that the membranous portion of the trachea is torn from its cartilaginous attachment, or so that the trachea by being sharply overstretched is fractured just above the carina. The mechanism apparently is similar to that of traumatic bronchial rupture.[17] Rupture of the trachea may be associated with bronchial rupture or tears in the wall of the esophagus.

DIAGNOSIS OF TRAUMATIC TRACHEAL RUPTURE

Traumatic tracheal rupture should be suspected whenever a sharp blow to the anterior chest or suprasternal region is followed by a cough and subcutaneous emphysema. With small tears, the cough will be hacking and nonproductive and the emergence of subcutaneous emphysema is delayed. Large lacerations cause severe cough with profuse hemoptysis and prompt massive mediastinal and cervical emphysema. Severe dyspnea and cyanosis are evident and usually unrelieved by oxygen therapy. As soon as the patient reaches the hospital, a tracheostomy should be done. Often tears in the cervical trachea will be discovered at this time and can be immediately repaired. If the mode of injury makes intrathoracic rupture more likely, the patient should be taken to the operating room where a bronchoscopic examination should be done to determine the extent and location of the tear (trachea or bronchi) as a preliminary to an immediate thoracotomy for definitive repair.

CLASSIFICATION

From the standpoint of treatment, tracheal rupture can be conveniently divided into three general classifications: (a) small tears which require only tracheostomy as definitive therapy, (b) large tears in the cervical trachea, and (c) large tears in the intrathoracic portion of the trachea which require immediate repair.

Small Tears.—Small tears of the trachea may result from penetration by sharp instruments, such as a knife blade or small caliber bullet, in which the skin incision heals quickly, thus, in effect, producing a closed injury. They also may occur secondary to sharply localized blows over the sternal region and sternal notch. The fact that the tear is small can be determined by the delayed emergence of a hacking cough and subcutaneous emphysema in the cervical region. Usually there is little or no expectoration of bloody mucus as contrasted with the profuse hemoptysis accompanying larger tears. Small tracheal tears should be treated by immediate tracheostomy which becomes the definitive therapy. The presence of the tracheostomy prevents the forcing of air through the tear when the patient coughs, since the patient cannot raise the intratracheal pressure by straining against a closed glottis. Small tears seal rapidly and the tracheostomy tube can usually be safely removed in 4 to 6 days.

The following 3 cases illustrate the problems presented by small tears.

CASE 1.—A pneumatic hammer, while in operation, slipped from the grasp of a 28-year-old laborer and struck him sharply above the sternal notch. He was temporarily dazed by the blow but recovered quickly. A few minutes later he reported to the emergency room of a hospital where he was examined and told that he had no serious injury. He was seen by the company physician approximately an hour after leaving the emergency room. By this time a hacking cough and swelling of the neck had developed. Palpation of the neck revealed the crepitus of subcutaneous emphysema. By the fifth day the subcutaneous emphysema had extended into the scalp, down the arms into the wrists, and down the abdomen and legs to the ankles. The patient was swollen beyond recognition and was in a semi-stuporous state. An immediate tracheostomy was done followed by prompt improvement. The tracheostomy tube was removed on the seventh day after its insertion and 2 days later the patient was discharged from the hospital. At this time the subcutaneous emphysema had almost completely absorbed.

CASE 2.—A 12-year-old boy was accidentally shot through the neck with a 22-caliber bullet. Very little bleeding occurred from the wounds of entrance and exit. The skin wounds promptly sealed. However, a hacking cough developed and the neck began to swell. The patient was admitted to the hospital immediately after the accidental wounding. Approximately 48 hours later the subcutaneous emphysema had spread alarmingly over the neck, chest, and shoulders. A tracheostomy was done. The subcutaneous emphysema rapidly absorbed and the tracheostomy tube was removed on the fifth day after its insertion. The patient left the hospital the following day and subsequently experienced no further difficulty.

CASE 3.—A 4-year-old girl while playing was struck on the upper anterior chest by an empty swing. She was knocked down but got up quickly and after crying for a few minutes went on playing. The accident occurred at approximately 11 A.M. Following the accident, a hacking cough developed and by 4 o'clock in the evening her neck was swollen. A physician recognized that the swelling was due to subcutaneous emphysema, and admitted her to a hospital for observation. A roentgenogram of the chest showed subcutaneous emphysema but no evidence of pneumothorax. During the subsequent 48 hours the emphysema slowly spread over the chest and abdomen and up into the face until the eyes were closed. By this time it was thought that her general condition was deteriorating. A rupture of the trachea was diagnosed and a tracheostomy was done. The tube was left in place 4 days. Recovery was rapid and complete.

Large Tears or Rupture-Separation of the Cervical Trachea-Acute.—Traumatic rupture of the cervical portion of the trachea should be suspected when,

after a severe blow to the region of the manubrium or sternal notch, severe dyspnea, cyanosis, and massive subcutaneous emphysema develop rapidly. A cervical incision should be made immediately upon the patient's admission to the emergency room of the hospital. If it is found that the tracheal tear has not completely disrupted the continuity of the trachea, the laceration can be sutured and a tracheostomy done in another area. The distal portion of the completely severed trachea retracts into the mediastinum due to the elasticity of the intercartilaginous ligaments. The finding of complete disruption of the trachea at the time of exposing the cervical trachea calls for immediate reanastomosis of the torn ends of the trachea followed by tracheostomy below the point of anastomosis. Beskin[1] first reported successfully treating by immediate repair a patient having a rupture-separation of the cervical trachea. The following case illustrates the management of such a situation.

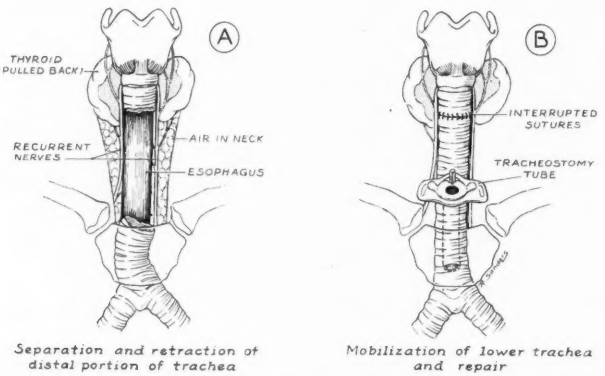

Fig. 1.—Case 4. Acute, closed, traumatic separation of cervical trachea in which the patient was treated with immediate suture and tracheostomy.

CASE 4.—A 32-year-old man, driving while intoxicated, had a head-on collision and suffered a steering-post injury to the lower neck and upper chest. On admission to a hospital, an intern recognized that because of the dyspnea, cyanosis, and massive subcutaneous emphysema, immediate tracheostomy was indicated. A vertical incision was made above the sternal notch. A considerable amount of air and bloody fluid was released but the trachea could not be immediately identified. Blind palpation with the finger revealed that the trachea had completely disrupted and the lower portion had retracted into the mediastinum. The intern was able to grasp the lower end of the trachea with an Allis clamp and draw it up into the incision. Bloody secretions were aspirated from the tracheobronchial tree and oxygen was insufflated into the lower trachea. He then called for the resident in thoracic surgery, and the patient was taken to the operating room. Here, under no anesthesia other than the drunken stupor, the resident surgeon* was able to carry out an accurate anastomosis of the trachea (Fig. 1) using fine braided wire sutures. A tracheostomy opening was placed 3 rings below the region of the anastomosis to aid with tracheobronchial aspirations and to protect the anastomosis during the period of healing.

The patient recovered quickly. It was possible to remove the tracheostomy tube 7 days after its insertion. The patient was discharged from the hospital with a normal voice. One

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month later, because of the persistence of a small sinus in the incision, a wire suture was removed from the anterior wall of the trachea through the bronchoscope. The region of the anastomosis was seen to be well healed with no significant narrowing of the tracheal lumen.

Chronic Sequelae of Traumatic Rupture of the Cervical Trachea.—The importance of definitive therapy at the time of the handling of the acute rupture of the trachea is emphasized by the severe problems encountered in restoring an airway when stenosis develops following tracheal rupture in the cervical region. Failure to bring about an immediate restoration of a normal airway results in chronic invalidism, severe mental trauma, multiple surgical procedures, and eventual loss of normal voice, either partially or completely. Nevertheless, an adequate airway can be established and patients should not be advised to accept a permanent tracheostomy as a solution to their problem. The problems presented by stenosis following traumatic rupture of the trachea in the cervical region are illustrated by the 5 cases which follow.

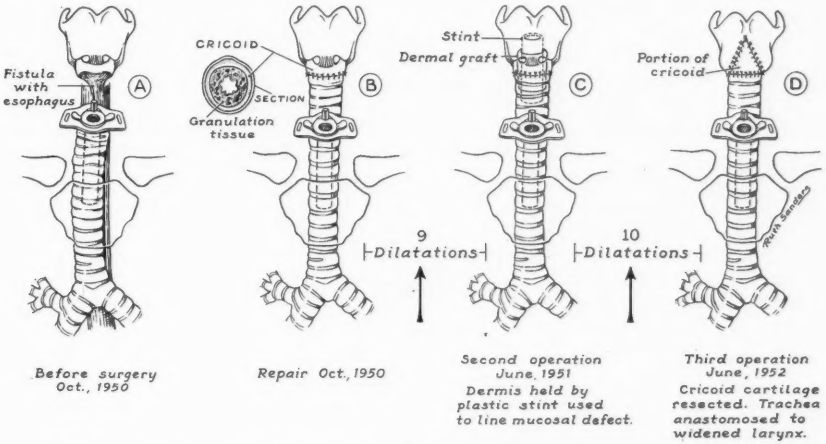

Fig. 2.—Case 5. Closed traumatic separation of cervical trachea (August, 1950) which resulted in tracheoesophageal fistula and subglottic stenosis. Multiple procedures were used before excision of the cricoid cartilage and anastomosis of the trachea to the widened larynx finally provided an adequate airway.

CASE 5.—On Aug. 7, 1950, an 11-year-old boy, while riding a horse, was struck by a wire clothes line just below the trachea. Immediate dyspnea occurred associated with hemoptysis, cyanosis, and subcutaneous emphysema. He was rushed to a hospital in a rural community. A surgical consultant from a neighboring city was called and recognized that immediate tracheostomy was indicated. When the neck was opened it was found that the cervical trachea had been completely torn away from the cricoid cartilage and had retracted into the upper mediastinum. The trachea was drawn up into the neck and a tracheostomy tube put in place. Since the patient's condition at this time was desperate, no attempt was made to do a definitive repair. On Aug. 25, 1950, he was admitted to Baylor University Medical Center. On this same day, a bronchoscopy was done under general anesthesia. It was found that a 4 mm. bronchoscope could be passed through the larynx to the region of the false cords. Here, ulcerated mucosa and exudate was encountered. The bronchoscope, however, could be passed through a fascial channel into the lower portion of the trachea. Because of inexperience with this type of injury it was thought that restoration of airway could be

accomplished by repeated dilatations. A subsequent dilatation was carried out on September 8. However, on October 19, it was found that the bronchoscope could be passed directly into the esophagus just beyond the cricoid cartilage indicating that a tracheoesophageal fistula was present. It was obvious that surgical excision of the stenotic area with closure of the esophagus and anastomosis of the trachea would be necessary (Fig. 2).

On Oct. 24, 1950, the region of the cricoid cartilage was dissected away from the upper esophagus and the opening in the esophagus closed. The lower trachea was dissected free from cervical structures, and the tracheostomy tube placed at a lower site. After cutting away the scarred ends of the trachea, an anastomosis was made between the lower trachea and the cricoid cartilage, using No. 34 stainless steel wire.

All incisions healed well. It was found, however, that an adequate airway through the region of the cricoid cartilage had not been established. During the next 7 months the tracheal stenosis was dilated on nine different occasions. Because of the failure to maintain an adequate opening through the trachea, it was decided that additional surgical repair would be required.

On June 6, 1951, the scar within the cricoid cartilage was excised by sharp dissection. A dermal graft was then laid within the cricoid cartilage and sutured to the tracheal and laryngeal mucosa with fine cotton sutures. A stent was placed within the larynx to maintain gentle pressure on the graft. The tracheostomy tube was kept in place.

On June 22, 1951, the plastic stent was removed and it seemed that an adequate airway had been established. A month later, however, it was found impossible to remove the tracheostomy tube because of an inadequate airway through the region of the cricoid cartilage. Again dilatations were attempted at intervals. After the tenth dilatation, which was carried out on April 25, 1952, it was obvious that an adequate airway could not be maintained.

On June 24, 1952, re-operation was carried out. At this time the trachea was cut across just below the region of the scarring. It could be seen that the stenosis lay within the cricoid cartilage. The cricoid cartilage was completely excised. The mucosa was trimmed back to normal tracheal mucosa. The thyroid cartilage was incised anteriorly. The laryngeal mucosa was normal just below the ventricles of the larynx. The lower trachea was anastomosed to the thyroid cartilage, leaving a wedge-shaped defect in the anterior portion of the cartilage. This was bridged by inserting a portion of the cricoid cartilage which had been excised. The cervical incision was closed.

The reconstructed airway healed well. It was not possible, however, to remove the tracheostomy tube because of the bilateral vocal cord paralysis. Bronchoscopic examination performed on June 15, 1953, indicated an adequate airway except for the narrow opening between the opposed vocal cords. Subsequently an arytenoid fixation was done which restored an adequate airway at the level of the vocal cords. Later the tracheostomy tube was removed and this patient, although unable to phonate normally, is now leading an active normal life.

CASE 6.—(Previously reported by Longmire.[11]) On Oct. 18, 1951, a 16-year-old boy came to the office wearing a tracheostomy tube. Eight years previously the cervical trachea had been avulsed from the cricoid cartilage by the handle of a car door. As an emergency procedure the associated lacerations of the esophagus and skin were repaired and the lower retracted portion of the trachea was brought up to the suprasternal notch to serve as a tracheostomy.

Two years later at another institution an attempt was made to bridge the 3 cm. gap between the normal larynx and the retracted lower end of the cervical trachea. At that time it did not seem feasible to re-anastomose the trachea to the cricoid cartilage. An acrylic tube was prepared which was used externally to bridge the gap from the larynx to the tracheostomy opening.

A year later at the same institution a Lucite tube was constructed which passed from the larynx to the tracheostomy opening through a subcutaneous tunnel. It was hoped that at a later date sufficient rigidity would occur so that the Lucite tube could be removed. When

this was attempted a year and a half later it was found that the unsupported channel was not rigid enough to support an adequate airway, necessitating reinsertion of the Lucite tube.

When first seen by the authors in October, 1951, the Lucite tube was still in place (Fig. 3). The airway, however, between the larynx and the retracted trachea was not sufficient to permit adequate respiration and the boy had lived in constant terror of not being able to breathe because of the necessity of having to rely on the tracheostomy tube. Both vocal cords were paralyzed and, in addition, his breath was foul due to suppuration around the Lucite tube.

Although repair was advised the patient demurred until 1953, when he accepted a program of attempted restoration of the laryngotracheal airway. Preliminary arytenoid fixation was done to widen the airway at the level of the vocal cords. On July 23, 1953, the Lucite tube was removed and its scarred channel excised. The cervical portion of the trachea which had retracted into the mediastinum was dissected free and the tracheostomy was moved to a lower level. In freeing up the larynx and cricoid cartilages, it was found that one intact tracheal ring was still present below the cricoid cartilage. It was not possible to do an end-to-end anastomosis of the trachea except under extreme tension. A dermal graft, reinforced with stainless steel wire mesh, was then fashioned into a tube and sutured to the ends of the trachea to bridge the gap. Immediate healing after operation seemed satisfactory.

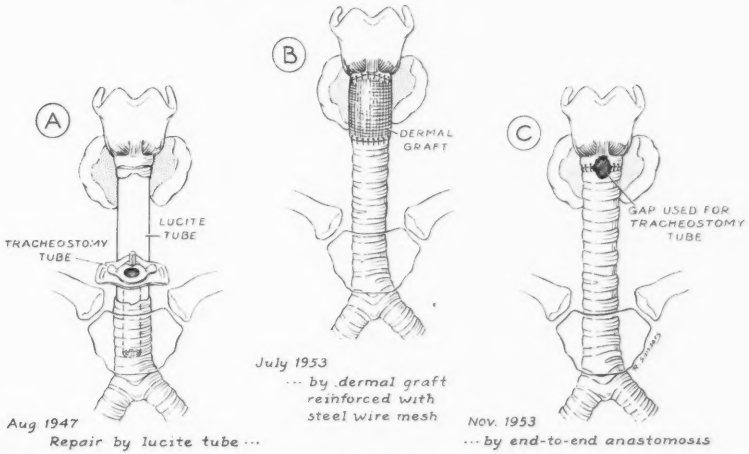

Fig. 3.—Case 6. Traumatic fracture of trachea (injury, 1944). Chronic phase of rupture separation of the cervical trachea in which the patient had been previously treated with a subcutaneous Lucite tube in an attempt to establish an airway. A staged procedure with lengthening of the intercartilaginous ligaments allowed restoration of the tracheal airway by end-to-end anastomosis.

Over a period of weeks, the size of the tracheostomy tube was gradually reduced to a No. 2 tracheostomy tube and, finally, 2 months after plastic repair, the tracheostomy tube was removed. A month later, however, the boy was having difficulty with respiration and a bronchoscopic examination revealed granulation tissue at the site of the anastomosis at the lower end of the graft. A tracheostomy tube was reintroduced. It was realized at this time that an additional procedure would be necessary to obtain an adequate airway.

On Nov. 23, 1953, the patient was again operated upon. The wire-reinforced dermal graft was removed. It was found that during the interim the intercartilaginous ligaments had stretched sufficiently to permit an end-to-end anastomosis of the trachea, without tension, using fine wire sutures. A small gap was left in the anastomosis anteriorly to permit insertion of a No. 2 tracheostomy tube. Wound healing was satisfactory. Within a week it was possible to remove the tracheostomy tube. Subsequently the patient has been able to breathe without difficulty, although there is almost complete loss of phonation.

CASE 7.—On Oct. 14, 1955, a 15-year-old boy rode a motor scooter under a wire cable that was used to mark space for parking. He immediately had a feeling of suffocation and strangulation. Since there was a laceration of the skin of the neck, subcutaneous emphysema did not result. He was seen almost immediately by a physician who inserted a tracheostomy tube into the lower portion of the torn cervical trachea and repaired the laceration of the skin. On Oct. 25, 1955, he was admitted to Baylor University Medical Center for definitive repair of the disrupted trachea and salivary fistula which had resulted from the injury. On October 26, the lower end of the trachea was dissected free and a new tracheostomy opening made at a lower level. The upper portion of the torn trachea was then dissected free. It was found that 3 cartilaginous rings remained below the cricoid cartilage. Using stainless steel wire in the cartilage and 5-0 silk in the mucosa, an anastomosis was then made between the 2 torn ends of the trachea. Skin closure was then left incomplete to allow for draining of the salivary fistula.

Wound healing, except for the fistulous site, was good. The tracheostomy tube was removed on the fourth postoperative day.

A bronchoscopic examination made on November 15 revealed that the right vocal cord moved normally but that there was paralysis of the left vocal cord. The tracheal anastomosis was well healed with no narrowing of the lumen.

The salivary fistula subsequently healed and this patient now has a normal airway but does have a hoarse voice due to injury to the left recurrent laryngeal nerve, occurring either at the time of the accident or during the dissection necessary for the definitive repair of the disrupted trachea.

CASE 8.—A 24-year-old man was injured Nov. 7, 1958, sustaining a crushing injury to his neck when he struck a platform while operating a dirt loader. An emergency high tracheostomy was done because of tracheal obstruction. On Dec. 9, 1958, the tracheostomy was lowered and a 9 mm. polyethylene tube was placed through the area of injury. Portions of the cricoid cartilage and the first two tracheal cartilages were missing and this area was involved in a fibrous scar. In February, 1959, the scar tissue was excised through a midline incision in the trachea and a dermal graft supported by sponge rubber was put in place. Infection supervened and the dermal graft was lost. An 11 mm. polyethylene tube was later placed through the stenotic area.

The patient was then referred to the authors. On Aug. 22, 1959, the stenotic area in the trachea was excised and the trachea anastomosed to the remainder of the cricoid cartilage posteriorly and the thyroid cartilage anteriorly, using No. 32 steel wire sutures. A stenosis again developed which necessitated a second operation April 23, 1960, at which time the scarred stenotic area was excised and an anastomosis performed between the trachea and the thyroid cartilage, with the use of 3-0 Merselene sutures. The thyroid cartilage was almost completely divided in a vertical direction and wedged open by means of a piece of hyoid bone sutured in place by fine wire sutures. The remainder of the anastomosis was completed by suture of the trachea over the lower portion of the transplanted hyoid bone.

Complete healing with an adequate lumen occurred and 7 days later the tracheostomy tube was removed. This patient has maintained an adequate airway.

CASE 9.—A 67-year-old woman had noticed progressive respiratory difficulty for approximately 15 years after an accident in which she had been struck sharply across the neck by a steel wire. She was admitted to a hospital where, at the time of bronchoscopic examination, it was found that there was stenosis of the trachea approximately 1 cm. below the vocal cords. Tracheostomy was done which gave marked relief from the difficult breathing. Because of staphylococcus infection around the tracheostomy tube, surgical repair of the stenosis was delayed. On Jan. 26, 1960, the stenotic portion of the trachea which involved the two upper cartilaginous rings was excised. Scar tissue within the cricoid cartilage was removed. The cervical trachea was then anastomosed to the cricoid cartilage with no attempt to approximate the mucosa of the trachea and larynx. The incisions healed and the tracheostomy tube was removed.

The tracheostomy tube was reinserted as an emergency measure in another city on March 31, 1960. Bronchoscopic examination revealed that the lumen at the level of the cricoid was choked with granulation tissue. Repeated bronchoscopies, at which time sutures and granulation tissue were removed, failed to re-establish a lumen that would allow removal of the tracheostomy tube.

The patient was referred to the authors and admitted to Baylor University Medical Center on March 6, 1961. Bronchoscopic examination on March 7, 1961, showed fibrous stenosis at the level of the cricoid cartilage that would admit a 4 mm. bronchoscope. On March 10 the cricoid cartilage was resected and the trachea anastomosed to the incised thyroid cartilage, using fine silk for the mucosa and No. 34 stainless steel wire in the cartilage. Wound healing was complicated by a staphylococcus infection which is subsiding. It is too early to determine whether or not the tracheostomy tube can be removed without additional surgery.

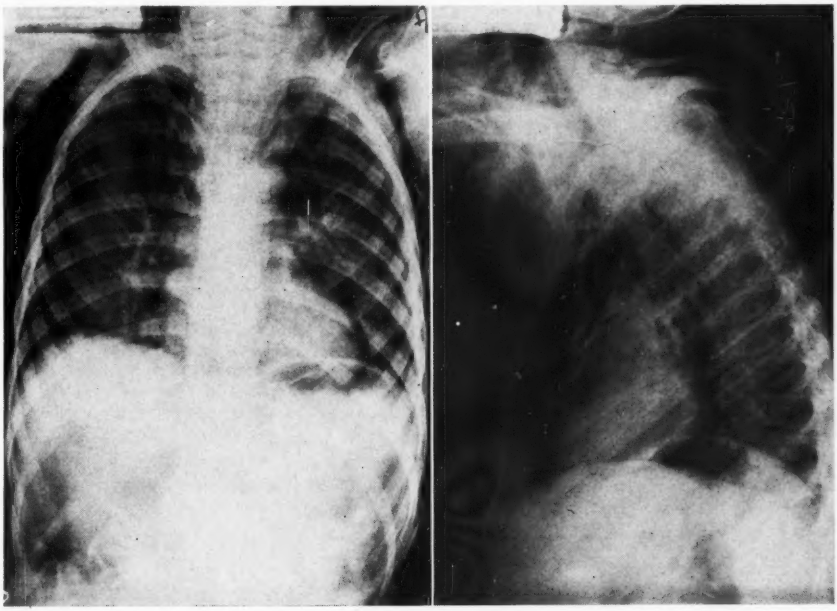

Fig. 4.—Case 10. Roentgenogram of the chest of a 5-year-old boy who sustained a long laceration of the membranous portion of the thoracic trachea. Note massive subcutaneous and mediastinal emphysema with no evidence of pneumothorax or rib fractures.

Intrathoracic Tears of the Trachea.—Usually the presence of a large intrathoracic traumatic tracheal rupture is promptly apparent when, after a sharp blow to the anterior chest, dyspnea, cyanosis, massive subcutaneous emphysema in the cervical region, and profuse hemoptyses quickly develop. There may or may not be associated pneumothorax or fracture of ribs. Such an injury may produce bronchial or tracheal rupture and is best treated by immediate bronchoscopic examination and thoracotomy,[6, 14, 15] unless some associated injury, such as a severe head injury, makes thoracotomy inadvisable. If thoracotomy is inadvisable, an immediate tracheostomy should be done to minimize the effect

of the tracheal rupture and to facilitate aspiration of bloody secretions from the tracheobronchial tree. The ideal management of a large intrathoracic tracheal rupture is illustrated by the following cases.

CASE 10.—A 5-year-old boy, while swinging, was struck sharply across the chest by the horizontal bar of the swing's support when the unanchored swing structure fell to the ground. He was taken immediately to the local hospital where it was noted that he already was cyanotic, was becoming alarmingly short of breath, and was coughing up blood. Massive subcutaneous emphysema of the cervical region developed and spread over the chest and up into the face. He was rushed by ambulance to Baylor University Medical Center. Here a roentgenogram of the chest revealed severe mediastinal and subcutaneous emphysema but no pneumothorax or rib fractures (Fig. 4). He was taken immediately to the operating room. A bronchoscopic examination was done under general anesthesia and the operating room prepared for surgery. When the bronchoscope was introduced into the trachea, blood

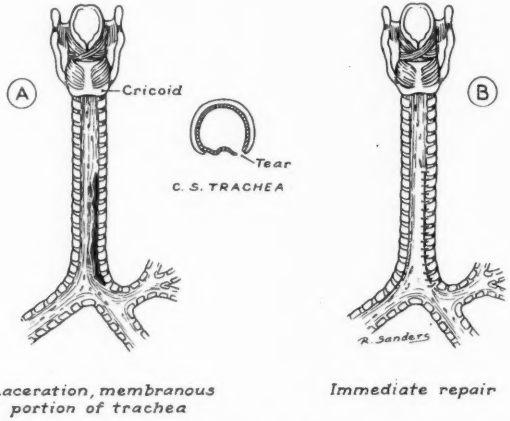

Fig. 5.—Case 10. Closed traumatic tear of trachea. Long laceration in the membranous portion of the intrathoracic trachea in which the patient was treated by right thoracotomy and suture of laceration. Recovery was prompt and complete.

was encountered and aspirated. The bronchoscope was passed down to the bifurcation of the trachea, where a tear in the membranous portion of the trachea was seen which started in the first portion of the right main bronchus. As the scope was withdrawn, it was determined that the linear laceration extended along the right posterior wall of the trachea at the point where the membranous portion joined the cartilaginous rings extending throughout the entire intrathoracic portion of the trachea. After bronchoscopy an intratracheal tube was put in place and the patient turned onto the left side. A right parascapular incision was made rapidly and the pleural cavity opened through the fifth intercostal muscle bundle. As soon as the posterior mediastinal pleura was opened, the long tear in the right wall of the trachea and right bronchus was apparent. The laceration was repaired with the use of interrupted cotton sutures (Fig. 5). After instituting pleural drainage to a water-seal bottle the thoracotomy incision was closed. The patient was turned onto his back and, before the intratracheal tube was removed, a tracheostomy was done.

The patient's postoperative course was uncomplicated. The subcutaneous emphysema was rapidly absorbed. The tracheostomy tube was removed on the fourth postoperative day and the patient was discharged to his home on the sixth postoperative day with all incisions healed. Thus, rapid surgical interference with restoration of a normal air passage allowed quick recovery with no sequela.

CASE 11.—A 5-year-old boy, on April 6, 1956, in an identical type of accident with a swing as in Case 10, sustained a small linear laceration of the membranous portion of the intrathoracic trachea just above the carina. At the same time the left main bronchus was completely disrupted. The tracheal laceration healed without repair. Six weeks after the injury, a left bronchial anastomosis was done through a left thoracotomy which resulted in restoration of the airway to the left lung. The child recovered without sequelae. Hood and Sloan[9] have reported a very similar small tracheal tear that healed spontaneously.

Intrathoracic Rupture-Separation of the Trachea.—An intrathoracic tracheal rupture-separation recognized late after tracheal stenosis has occurred results in a more formidable surgical problem for correction than one which can be repaired during the acute stage. Richards and Cohn[16] have stressed the extreme difficulties of the late management of this type of tracheal injury. The development of the stenosis is associated with marked scarring of the mediastinum surrounding the trachea because of the mediastinal abscess that inevitably follows rupture of the trachea. An esophageal tear may be associated and a fistula result. During the post-traumatic period the use of a long tracheostomy tube will be necessary in order to maintain an airway. As long as the tracheostomy tube is in place the patient feels secure but is unable to phonate. Attempts to increase the lumen of the trachea at the point of stenosis by repeated dilatations are futile because of the loss of cartilaginous support and malalignment of the severed ends. Resection of the stenotic portion of the trachea with anastomosis of the separated ends is the only means of achieving restoration of the airway and subsequent removal of the tracheostomy tube. The problems presented by post-traumatic stenosis of the intrathoracic trachea are illustrated by the following 2 cases.

CASE 12.—A 33-year-old man was injured on Nov. 30, 1957, in a head-on collision while driving a car. A roentgenogram taken of the chest upon admission to a hospital showed bilateral pneumothorax and massive subcutaneous emphysema but no evidence of fractured ribs (Fig. 6). Intercostal drainage was instituted on the right side to re-expand the lung. In addition to the chest injury there was a compound fracture of the left ankle involving the tibia, fibula, and tarsal bones. Sixteen days after injury the patient was allowed to return to his home, although at this time he had a severe harassing cough and was short of breath. Soon after his return home the dyspnea became so severe that he was rushed to a hospital where a tracheostomy was done. Since this did not relieve the dyspnea, a filiform catheter was threaded down the trachea and respiration was supported by insufflation of oxygen through this catheter. The patient was then returned to the hospital where the initial injuries had been treated. Tracheoscopy revealed that there was a severe stenosis, approximately 2 cm. above the bifurcation of the trachea. By dilatations, the stenotic area was gradually enlarged so that a long No. 9 tracheostomy tube could be placed through the region of stenosis.

The patient was referred to the authors for additional treatment and was admitted to Baylor University Medical Center on Oct. 6, 1958. He was still wearing the long No. 9 tracheostomy tube. The following day, a right thoracotomy was done. The lower trachea, including the area of stenosis, was completely dissected free from the neighboring mediastinal structures. It was found that the tear through the trachea was not complete but that approximately one sixth of the diameter of the trachea was intact along the left lateral wall (Fig. 7). The tracheal scar was excised. The cartilaginous portion of the trachea was reanastomosed using No. 30 stainless steel wire. The membranous portion of the trachea was reconstructed using No. 30 cotton sutures. Pleural drainage to a water-seal bottle was established through an intercostal space and the thoracotomy incision was closed.

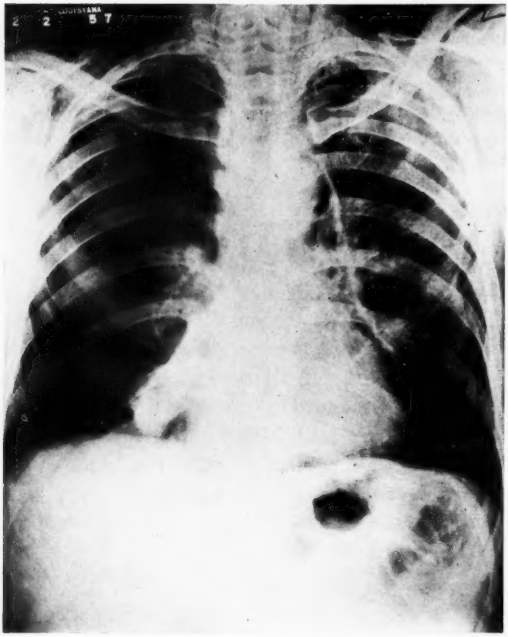

Fig. 6.—Case 12. Intrathoracic rupture-separation of the trachea. Note bilateral pneumothorax and cervical and mediastinal subcutaneous emphysema but no evidence of fractured ribs. The outline of the tracheal air column can be seen revealing partial disruption of the trachea, 2 cm. above its bifurcation.

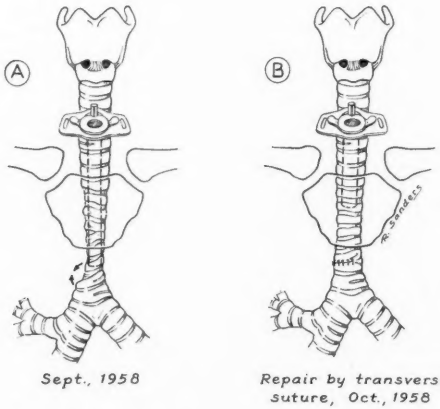

Fig. 7.—Case 12. Closed traumatic rupture of the intrathoracic trachea (November, 1957) which resulted in stenosis. Repair by transverse suture resulted in restoration of an adequate tracheal airway.

The postoperative course was complicated by the formation of a clotted hemothorax which subsequently became infected and required continued drainage. Since a good airway had been established it was possible to remove the tracheostomy tube on the fourth postoperative day and the tracheostomy stoma slowly closed. The pleural drainage tube was removed on Dec. 29. The patient was last seen for examination on Jan. 13, 1959. There was no impairment of breathing. His voice was normal. X-ray studies of the chest showed only slight narrowing of the trachea at the point of traumatic rupture and repair.

CASE 13.—On July 21, 1959, an 18-year-old boy, while driving a car, struck a steel bridge abutment and sustained a steering-post injury to the chest. He was taken immediately to a hospital where, upon admission, he had massive subcutaneous emphysema associated with a cough which was productive of copious amounts of blood. Roentgenograms of the chest showed no evidence of fractured ribs. There were no important associated injuries. Two days after the injury, a right pneumothorax was recognized and treated with an intercostal

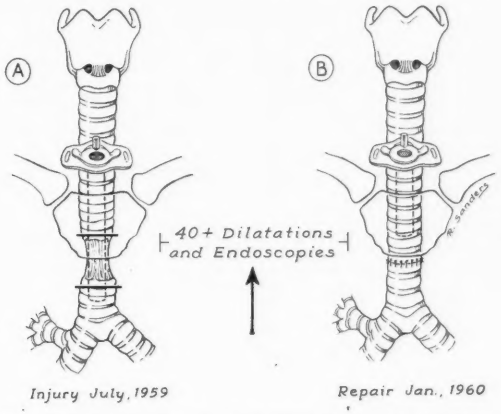

Fig. 8.—Case 13. Closed traumatic rupture-separation of the intrathoracic trachea resulting in severe stenosis. The patient was treated unsuccessfully by multiple dilatations. Repair by excision of scar tissue and anastomosis of the trachea established an adequate tracheal lumen.

tube connected to a water-sealed system. The right lung promptly re-expanded and the intercostal tube was removed. The cough persisted and was present at the time of his discharge from the hospital 10 days after his injury. While at home he soon developed severe shortness of breath marked by cyanotic episodes. He was hospitalized on three occasions before he finally became critically ill. On readmission to the hospital an emergency tracheostomy was done. At this time it was recognized that a tracheal stenosis had developed approximately 3 cm. above the bifurcation of the trachea due to traumatic tracheal rupture. Over a period of 40 days, dilatations of the stenotic area were carried out with the use of steel dilators through the tracheostomy opening. The patient was discharged from the hospital with the tracheostomy tube in place. Later he was seen by an endoscopist who examined the region of the stenosis and on several occasions removed granulation tissue. Finally a long No. 9 tracheostomy tube was put in place so that it passed through the region of the stenosis as a stent in the hope that epithelization of the stenotic area would allow removal of this tube later. Attempts to remove the tracheostomy tube, however, resulted in severe dyspnea.

The patient was seen by the authors and admitted to Baylor University Medical Center on Jan. 1, 1960, a little more than 5 months after the original injury. The following day a right thoracotomy was done. After tedious dissection, the lower trachea was dissected free from mediastinal structures. It was found that the trachea had separated approximately 3 cm. at the point of the rupture. The scarred ends of the trachea were excised and an end-to-end anastomosis was carried out, using No. 30 cotton sutures (Fig. 8).

The immediate postoperative recovery was uncomplicated. The tracheostomy tube was removed on the fourth postoperative day and the tracheostomy stoma closed completely in one week. The patient was discharged from the hospital on the fourteenth postoperative day.

On January 27 the patient was readmitted to the hospital complaining of wheezing respirations and dyspnea. The next day a bronchoscopic examination was done under general anesthesia. Granulation tissue had developed at the anastomotic site and partially obstructed the lumen. With a grasping forceps the granulation tissue along with eight cotton sutures were removed. This established a lumen at the region of the anastomosis, judged to be at least 15 mm. in diameter.

On February 17, a second bronchoscopic examination was done. Three additional cotton sutures were removed from the right wall of the trachea. The region of anastomosis had healed well, being epithelized except for the point where the cotton sutures lay exposed. There was some stenosis at the point of the anastomosis but the lumen appeared adequate for free respiration. The patient has continued to do well more than a year after repair of the trachea.

DISCUSSION

Thirteen cases of traumatic tracheal rupture have been presented, illustrating the ease of repair and the immediate good results obtained in the acute stage and the problems encountered with restoration of an airway and normally functioning larynx during the chronic phase. Nine patients had injury to the cervical trachea, and, in 4 patients, the intrathoracic portion was ruptured. Table I summarizes the cases and the results obtained. Three patients who had small tears of the cervical trachea recovered promptly after tracheostomy. One patient with rupture-separation of the cervical trachea who was treated by immediate suture and tracheostomy completely recovered without loss of voice. All 5 patients who developed stenoses after rupture-separation of the cervical trachea had multiple surgical procedures. Following excision of the scarred stenotic area and anastomosis, 4 patients now have an adequate airway without tracheostomy but have partial or complete loss of normal phonation. The other patient has been operated upon too recently to be evaluated. One patient with a long tear of the intrathoracic trachea recovered promptly and completely after thoracotomy with suture of the laceration. In a patient with a smaller intrathoracic tracheal tear associated with rupture-separation of the left main bronchus, the tracheal tear healed spontaneously. Two patients with stenoses resulting from late recognition of rupture-separation of the intrathoracic trachea now have an adequate airway following excision of the scarred stenotic area and anastomosis of the trachea.

Partial or complete transverse rupture of the trachea leads to stenosis unless prompt primary repair is carried out. Various methods of treatment have been advocated for tracheal stenosis. These include multiple gradual dilatations, use of plastic and metal prostheses to maintain the size of the lumen, and excision of the scar in the stenotic area followed by application of skin grafts to the denuded air passage which were held in place temporarily by plastic molds or stents. Articles by Som,[18] Hanckel,[7] Harkins,[8] Erich,[5] Nach and Rothman,[13] Mitchel and Davis,[12] Cummings,[3] and Longmire[11] have all dealt with the problems of reconstruction of the airway following development of stenoses. In

TABLE I. TRAUMATIC TRACHEAL RUPTURE

TYPE	NUMBER OF CASES	TREATMENT	COMPLICATIONS OR SEQUELAE	RESULT
Small tears and perforations	3	Tracheostomy	None	Complete recovery
Rupture-separation, cervical, acute	1	Immediate repair, tracheostomy	None	Complete recovery
Rupture-separation, cervical, delayed, with stenosis	5	Plastic repair, tracheostomy	Loss of normal voice, 5 Staphylococcus infection, 1	Adequate airway, 4 Inadequate airway, 1
Intrathoracic tear, small, with left bronchial rupture	1	Repair bronchial rupture	None	Complete recovery
Intrathoracic tear, large	1	Immediate repair, tracheostomy	None	Complete recovery
Rupture-separation, intrathoracic; stenosis	2	Tracheostomy, delayed anastomosis	Hemothorax, 1	Adequate airway
Total	13			

some instances, dermal grafts lining the inner trachea and larynx have allowed establishment of an adequate airway so that permanent tracheostomy was not required. Generally the use of plastic substitutes for the trachea has been disappointing. The complete excision of scar tissue and adequate mobilization of the trachea, with accurate approximation of the mucosa and cartilaginous rings, seem to be the procedure of choice. High stenosis at the level of the cricoid, as a result of tracheal rupture, may be treated by excision of the cricoid cartilage and anastomosis of the trachea to the thyroid cartilage which has been incised and wedged open by the insertion of a portion of the excised cricoid cartilage or hyoid bone. In traumatic tracheal rupture there is usually little loss of tracheal substance so that end-to-end anastomosis should be feasible even in patients having old injuries with separation of the tracheal segments.

SUMMARY

1. Thirteen cases of traumatic tracheal rupture are reported. Nine involved the cervical and 4 the intrathoracic trachea.

2. Primary repair of the torn trachea during the acute phase is the treatment of choice and should be accomplished whenever possible.

3. The disadvantages and technical problems encountered in secondary repair of the ruptured trachea are discussed.

4. Loss of normal voice is the most important sequela of delayed repair of rupture of the cervical trachea.

5. Restoration of an adequate airway by excision of scar tissue and accurate anastomosis of the trachea, without resort to permanent tracheostomy and plastic prostheses, should be feasible in all cases of stenosis as a result of traumatic tracheal rupture.

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DISCUSSION

DR. R. MAURICE HOOD, Austin, Texas.—Since Dr. Sloan and I reported our series and reviewed this problem 2 years ago, I have had two additional cases of injury to the trachea, in both of which there was concurrent esophageal laceration. These will not be reported in detail but there are two points that I would like to make: the physical findings in these patients do not necessarily reflect the extent of the tracheal injury and it is not at all unusual to find major tracheal or bronchial obstruction without any symptoms at the time of injury that indicate that the airway system has been injured. Only the late findings of atelectasis and tracheal and bronchial stenosis will indicate this. Although the tracheal injury is more likely to produce severe emphysema and airway obstruction than the bronchial injuries, nevertheless it may not. Aggressive maneuvers may be required in order to maintain life in some of these patients.

Certainly, it is always desirable to repair these at the earliest stage and the results of primary repair are usually excellent, as Dr. Shaw has shown, and the results of late repair are usually not quite as satisfactory.

However, lest surgeons be misled into operating upon all these patients as emergencies, it is quite within reason to stabilize the patient's cardiorespiratory system with tracheostomy and interpleural catheters so that primary repair may be done under ideal circumstances, not as emergency procedures by untrained personnel and under circumstances that are less than ideal. This primary repair may be delayed even for several days without harm to the patient and with the desired good result.

DR. DAVID H. WATERMAN, Knoxville, Tenn.—Our experience with tracheal rupture has been similar to that of Dr. Shaw, although in a somewhat smaller series of cases. I would like particularly to emphasize one point made by Dr. Shaw, namely, the importance of establishing an immediate airway. Bronchoscopy certainly is the best way to do this.

In our last case of complete rupture-separation of the trachea, we erroneously thought that we could intubate the patient without preliminary bronchoscopy. While the anesthetist was unsuccessfully attempting to do this, cardiac arrest from anoxia occurred; this brought about the paradoxical situation of the thoracic surgeon intubating the patient with the bronchoscope while the anesthetist was performing emergency thoracotomy and cardiac massage. These reciprocal maneuvers, though somewhat bizarre, were fortunately successful.

This case, incidentally, was also unique in that the anesthetist, in his commendable haste to open the chest, introduced a new thoracotomy approach in thoracic surgery—an incision made at right angles to the ribs. We feel, however, that this innovation will not replace the customary incision.

When resection of a portion of the trachea has been necessary, our experience with certain artificial grafts has not been particularly rewarding. I am glad to learn that in such cases Dr. Shaw was able to reconstruct the trachea by bringing the ends together, as this certainly seems a preferable procedure.

DR. RICHARD H. SWEET, Boston, Mass.—This is a most important contribution because it brings up two basic principles which I think should be emphasized. The first is the importance of an aggressive attitude in the management of these cases. This viewpoint should be disseminated widely among thoracic surgeons and general surgeons as well, namely, that, within reason, these injuries should be treated promptly.

The second basic principle is the inadequacy of prosthetic devices in the attempt to restore continuity to the trachea. I would like to call your attention to the fact that the same difficulties arise in the esophagus. In both instances we are dealing with a contaminated structure. Dr. Shaw has stressed the importance of an accurate mucosal approximation. This should be obvious to us all, as experience with the esophagus has shown that any effort to restore continuity by such means would never work.

DR. SHAW (Closing).—I wish to thank Dr. Hood, Dr. Waterman, and Dr. Sweet for their remarks. I would like to talk briefly about the matter that Dr. Hood brought up concerning delay of the repair. Of course, immediate repair may not always be feasible because of conditions under which the patient is seen or, perhaps, because of an associated injury, such as a severe head injury, that would make a general anesthesia and thoracotomy inadvisable.

However, I believe that if delay is necessary, the matter of timing becomes rather important. Certainly a tracheostomy must be done and, of course, the tracheostomy tube, in case of a rupture-separation in the intrathoracic portion, will have to be long enough to go through the stenotic area that will inevitably result.

If there is delay, I think the delay should be months and not a week or so, because, inevitably, a mediastinal abscess develops at the point of tracheal rupture. This will be contiguous with the vena cava and the esophagus—both structures with which it may be hard to deal. If the operator moves in too soon before this abscess has cleared, an infected trachea will result which will not hold sutures well. In case of delay I think it would be preferable to put the tracheostomy tube in place, give antibiotic therapy, and wait for a suitable time until all signs of mediastinal infection have disappeared and there has been a complete resolution of suppuration to clean fibrosis. Although this will make the dissection difficult, it will be better than breaking into an abscess cavity.

THE SURGICAL MANAGEMENT OF METASTATIC NEOPLASMS IN THE LUNG

Earle W. Wilkins, Jr., M.D., John F. Burke, M.D. (by invitation), and John M. Head, M.D. (by invitation), Boston, Mass.

IN 1938, Dr. E. D. Churchill and Dr. J. D. Barney[2] published a report of one of the very early instances of removal of a metastatic renal cell carcinoma from the lung. The patient, a woman, lived for 23 years after a partial lobectomy in 1933 and died of coronary artery disease. This is, we believe, the longest recorded survival following extirpation of a pulmonary metastatic carcinoma. The success in this instance encouraged the excision of metastatic malignancies in the lung in subsequent patients.

During the past 28 years, 67 patients have undergone 75 operations for removal of pulmonary metastases at the Massachusetts General Hospital. One patient had three separate thoracotomies, 4 had bilateral operations, and, in 2, second procedures were done on the same side. The remaining 60 patients had one operation each. Patients were included from both the ward and private services, and, in the latter category were private patients of six different surgeons. There may have been, therefore, some differences in the criteria for operability. In general, patients were operated upon only if the primary neoplasm had been eradicated and no other evidence of metastasis was detectable. All operations were performed as possible curative procedures, none purely for palliation. Survival figures do reflect, among other factors, the criteria for selection of operative candidates.

Follow-up examination has been complete for all 67 patients. The status of each has been accurately determined as of Dec. 31, 1960.

ANALYSIS OF FINDINGS

Primary Site.—The wide variety of primary sites is listed in Table I. The miscellaneous category includes 2 cases of breast carcinoma, a synovioma, and one case each of thyroid, ovary, prostate, and stomach carcinoma.

Age and Sex (Fig. 1).—The peak incidence is in the sixth and seventh decades. This is not, however, a problem peculiar to advancing age. There were 11 patients under 40 years of age at the time of the pulmonary operation, 3

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Read at the Forty-first Annual Meeting of The American Association for Thoracic Surgery at Philadelphia, Pa., April 24-26, 1961.

TABLE I. PRIMARY SITES

Colon and rectum	17
Kidney	16
Connective tissue	7
Uterus	5
Melanoma	4
Bladder	4
Bone	4
Testis	3
Miscellaneous	7
Total	67

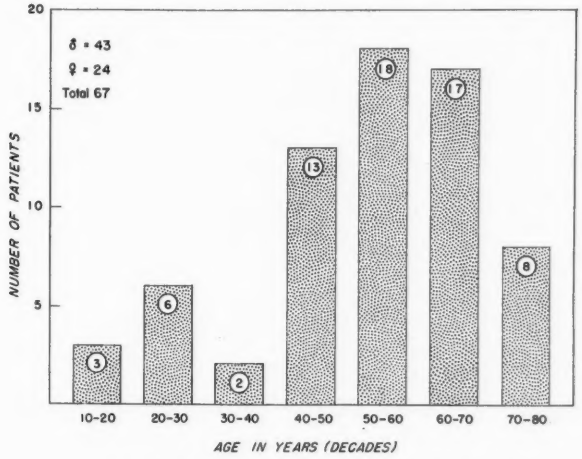

Fig. 1.—Distribution of patients by age and sex.

under 20. The youngest patient was 15 years old, a girl with metastatic osteogenic sarcoma. The oldest was a man of 79 with an adenocarcinoma of the sigmoid colon.

Symptoms.—Thirty of these patients had no symptoms attributable to their pulmonary metastases. The remainder had one or more of the following complaints, listed in order of decreasing frequency: cough, hemoptysis, chest pain, fever, dyspnea, hoarseness, wheeze, and osteoarthropathy. These are also the common symptoms of primary bronchogenic carcinoma.

Roentgenograms.—Preoperative roentgenographic evaluation of the patients in this series was generally thorough and included posteroanterior, lateral, and overexposed grid views of the chest, fluoroscopy, and a barium esophagogram. Laminagrams were often valuable in the estimate of additional metastatic nodules. A thorough search was made for metastases in other locations.

The location of the metastases was divided uniformly among the lobes of the lungs. When this could be determined from the operative reports, there were seventeen nodules in the right upper lobe, fifteen in the right middle lobe, seventeen in the right lower lobe, thirteen in the left upper lobe, and thirteen

in the left lower lobe. These figures include cases in which more than one lobe was involved and account for the high figure for the middle lobe. There was no evidence that lesions from the lower part of the body metastasize to the lower lobes, or those from the upper part to the upper lobes; nor was there any evidence for metastasis to the ipsilateral lung.

Diagnosis.—In only 5 cases was the diagnosis confirmed prior to thoracotomy. Bronchoscopic biopsies were positive in 2 patients. In 3 others, exfoliated cells in the sputum revealed the diagnosis. Although the diagnosis was reasonably certain in all 67 patients, it was only at the time of thoracotomy that confirmation was obtained in the remaining 62.

Operations.—The specific procedures are listed in Table II. In principle, conservative excision was practiced in this series, undoubtedly more so than in surgery for primary pulmonary carcinoma. Local removal by wedge excision was considered sufficient in 18 cases. With few exceptions no attempt was made to perform extensive mediastinal lymph node dissections. When grossly enlarged nodes were apparent, an effort was made to remove as many as possible.

TABLE II. OPERATIONS*

Pneumonectomy	12
Lobectomy	41
Segmental excision	4
Local excision	18
Total	75

*Seventy-five procedures were performed on 67 different patients.

Mortality and Morbidity (Table III).—Of the twelve complications, only three were sufficiently severe to prolong hospital stay. Each of these patients had an empyema, one of whom had a known bronchial stump leakage. This man had a persistent bronchopleurocutaneous fistula until his death from carcinoma 6 months later. The other 2 patients responded to rib resection and drainage of the empyema. These 67 patients remained in the hospital an average of 15 days following thoracotomy. There were no deaths.

TABLE III. COMPLICATIONS

Mortality: 75 Operations		0
Morbidity:		12
Pleural effusion	3	
Hemothorax	2	
Empyema	2	
Bronchopleural fistula	1	
Wound sepsis	1	
Collapse, lung	1	
Cardiac arrhythmia	1	
Aphasia, transitory	1	

Secondary Lymph Node Metastases.—A question persistently posed concerning pulmonary metastases has been the presence of secondary node metastases, or nodes that presumably become involved as matastases from the pulmonary disease. It has been stated often that these do not occur. In this series,

positive bronchial or mediastinal nodes were encountered in 9 cases. In 25 operations, nodes that proved to be negative were removed. In the remaining 41 operations, nodes were not mentioned in the reports from the pathologist. Presumably none was removed in these 41 cases. In specimens in which nodes were reported, 26 per cent were positive. If all specimens with unreported nodes actually did contain negative nodes, this figure could drop as low as 12 per cent.

Survival.—Of the 67 patients, 13 survived 5 or more years (Table IV). Six of these 13 have died subsequently. One died after 23 years, one after 11 years, 2 after 7 years, and 2 died 6 years after pulmonary resection. Four of the 6 died of cardiac illnesses without known malignant disease; 2 died with persistent cancer. Of the 7 patients still alive, one has evidence of residual disease in the other lung. The remaining 6 are free of disease after periods of 5 to 15 years following lung resection.

TABLE IV. FIVE-YEAR SURVIVORS

PRIMARY	RESECTIONS	SURVIVORS	PER CENT
Kidney	16	5	31
Colon and rectum	17	3	18
Uterus	5	3	60
Connective tissue	7	1	14
Bone	4	1	25

Of those patients who have undergone surgery during the past 5 years, 8 of 24 are alive.

These results have been calculated by the cumulative survival method (Table V).[18, 28] This method gives a curve typical of many malignant primary lesions. The greatest mortality occurs within the first 2 years and the curve becomes relatively flat thereafter. The most interesting feature of this curve appears when it is superimposed on the cumulative survival curve for primary carcinoma of the lung (Fig. 2). The latter is from the Massachusetts General

TABLE V. CUMULATIVE SURVIVAL: MASSACHUSETTES GENERAL HOSPITAL, 1933-1960

TIME PERIOD ENDING (YR.)	DEATHS	DROPPED FROM STUDY	PATIENTS ENTERING	PATIENTS AT RISK	PERIOD MORTALITY (%)	PERIOD SURVIVAL (%)	CUMULATIVE SURVIVAL (%)
½	14	3	67	65.5	21	79	79
1	13	1	50	49.5	26	74	58
1½	5	1	36	35.5	14	86	50
2	6	2	30	29	21	79	40
2½	2	0	22	22	5	95	38
3	0	0	20	20	0	100	38
3½	0	1	20	19.5	0	100	38
4	1	0	19	19	5	95	36
4½	5	0	18	18	28	72	26
5	0	0	13	13	0	100	26
10	4	6	13	10	40	60	16

Hospital figures of the period from 1933 through 1956.[4] The five-year cumulative survival for resected metastatic pulmonary lesions is 26 per cent; for resected primary carcinomas it is 28 per cent. If the curves are extended to 10 years, these figures become 16 per cent and 18 per cent.

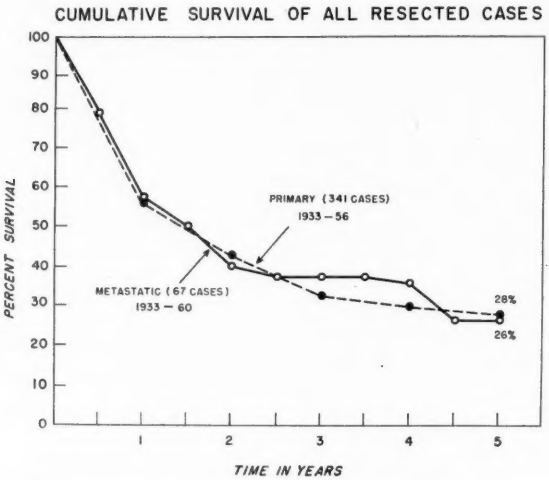

Fig. 2.—Comparison of results of excision of primary bronchogenic carcinoma and metastatic neoplasms in the lung.

CONTRIBUTORY FACTORS

It is interesting to consider various factors that may have some influence on survival after removal of pulmonary metastases. Seven factors may have some bearing.

1. *The Primary Lesion.*—In this series, carcinomas originating in kidney, large bowel, and uterus have had the best prognosis. Carcinomas arising in endocrine organs or in the breast have not given encouraging survival rates. Metastatic melanomas have yielded uniformly dismal results. Two patients in this series first demonstrated pulmonary metastases 15 and 17 years after eye enucleation for melanomas of the choroid. Melanoma of the eye can present very late evidence of pulmonary spread; when it does, it may be the first manifestation of disseminated disease.

In general, the presence of positive nodes in the specimen of the primary lesion is a bad prognostic omen. All of these patients, 4 with carcinoma of the rectum or colon and one with a carcinoma of the thyroid, died within 6 months of removal of the pulmonary metastasis except one who is alive and apparently free of disease after 3 years and 4 months. She had only one positive node in the specimen of the sigmoid resection.

2. *Secondary Lymph Node Metastases.*—Patients who have secondary node metastases present a poor prognosis. There were 9 of these patients. Four had primary lesions in the colon. Two had melanomas. There were single

instances of primary tumors in the urinary bladder, testis, and prostate. The patient who has survived the longest time was the one with primary carcinoma of the prostate. It is now 19 months since his pneumonectomy and mediastinal node dissection. He has also had bilateral orchiectomy and estrogen therapy, which may be very significant factors in his survival. The remaining 8 all died within 17 months.

3. *Time Interval Between Primary and Pulmonary Operations.*—In general, the greater the span of time, the better the possibility of long-term survival. Of 40 patients whose procedures were performed less than 5 years after the primary operations, 4 (or 10 per cent) survived 5 years after removal of the metastasis. Of 20 patients whose procedures were done more than 5 years after the primary operation, 8 (or 40 per cent) survived an additional 5 years. Seven patients had the metastasis removed first, before a primary was known, and only one of these is still alive.

4. *Extent of the Pulmonary Operation.*—If pneumonectomy was required to remove the metastatic disease, the outcome was usually poor. None of the 12 patients requiring pneumonectomy survived 5 years; only 2 survived 2 years. If pneumonectomy was not required, the type of lesser operation performed did not seem to influence the results. Following lobectomy, segmental excision, and local removal, 22 per cent, 25 per cent, and 17 per cent, respectively, survived 5 years.

5. *Number of Metastases.*—Multiplicity of metastatic nodules augurs poorly for survival. More than one nodule was found in six explorations. Actually, this was known preoperatively in 5 of the 6 cases. Two of these patients are alive after a period of more than 5 years. One is alive, with residual disease, 8 years after removal of two metastases from a carcinoma of the cecum; the other is free of known disease 6 years after removal of two metastases from a choriocarcinoma of the uterus. These metastases were located in the same hemithorax.

6. *Repeated Operations.*—This had a uniformly grave prognosis in this limited series. In 6 patients who had either a bilateral thoracotomy or a second exploration of the same hemithorax, death occurred from residual disease within 2 years. There is one exception. She is the patient who is alive 8 years after a right middle and upper lobectomy for two nodules. She required a lower lobectomy 5 years later for still a third metastasis and now has a nodule in the left lung.

7. *Bronchial Metastasis.*—There were only three such metastases in this series of 67 patients. It would not be wise, therefore, to attempt conclusions, but all 3 did badly. Two had primary lesions in the colon. The other had one that originated in the kidney. None survived 2 years.

SURVEY OF THE LITERATURE

Because this series of 67 patients is small and might be considered insignificant statistically, a review of the available literature was made. Whereas in 1947 Alexander and Haight[1] accumulated twenty-four instances of the removal

of metastatic disease from the lung, we have been able to find 355 cases.[3, 5-17 19-27, 29-34] Many other cases that have been reported were not included in this listing because of sketchy details, inconclusive diagnosis, or complete lack of follow-up information.

Unfortunately, follow-up data are incomplete in many of the 355 cases. Almost half have an unknown outcome less than 5 years after lung resection because of early reporting. Table VI shows an analysis of the types of primary carcinomas with known 5-year survivors. As in our series, results from renal, colonic, and uterine metastases are the best; those from breast and melanoma are poor. Further deductions are not reasonable from the data available.

TABLE VI. CARCINOMA* (SURVEY OF LITERATURE)

	NO. CASES	KNOWN 5 YR. SURVIVORS
Gastrointestinal	69	3
Urinary	47	6
Uterine	32	4
Mammary	31	2
Endocrine	31	3
Melanoma	17	0
Miscellaneous	27	0
Total	254	18

*The total numbers and favorable results from excision of metastatic carcinomas to the lung.

Table VII shows the results following removal of metastatic sarcomas. The miscellaneous group is composed largely of unspecified primary tumors. Patients with osteogenic sarcomas have done surprisingly well.

TABLE VII. SARCOMA* (SURVEY OF LITERATURE)

	NO. CASES	KNOWN 5 YR. SURVIVORS
Fibrosarcoma	32	3
Osteogenic	23	6
Synovioma	6	2
Chondrosarcoma	5	0
Neurogenic	4	2
Miscellaneous	31	3
Total	101	16

*The favorable results from excision of metastatic sarcomas to the lung.

There were twenty known operative deaths in the 355 cases of carcinoma and sarcoma, which is an incidence of 5.6 per cent. The total series, including the 67 in this presentation, amounts to 422 cases, with an operative mortality of 4.7 per cent.

TABLE VIII. CUMULATIVE SURVIVAL: SURVEY OF LITERATURE: CARCINOMA AND SARCOMA

TIME PERIOD ENDING (YR.)	DEATHS	DROPPED FROM STUDY	PATIENTS ENTERING	PATIENTS AT RISK	PERIOD MORTALITY (%)	PERIOD SURVIVAL (%)	CUMULATIVE SURVIVAL (%)
1	108	57	355	326.5	33	67	67
2	46	50	190	165	29	71	47
3	10	28	94	80	13	87	40
4	3	19	56	46.5	8	92	37
5	0	9	34	29.5	0	100	37

The 5-year cumulative survival for the 355 cases is 37 per cent, an unrealistically favorable figure due to the large number of patients with inconclusive follow-up information or early reporting (Table VIII). These figures do lend some measure of confirmation to the results in our own series.

CONCLUSIONS

1. Removal of pulmonary metastases in patients whose primary malignancy is otherwise controlled results in a 26 per cent 5-year cumulative survival. This is identical with the survival from primary bronchogenic carcinoma.

2. The most favorable results occur following excision of pulmonary metastases in patients whose primary lesions are osteosarcomas, or carcinomas originating in the colon, kidney, or uterus. Patients who have metastatic melanomas do poorly. Occasional long-term survivals occur after removal of pulmonary metastases which represent spread from a large variety of primary sites and cell types.

3. Factors contributing to an unfavorable prognosis include: involvement of lymph nodes in the specimen of the primary resection, positive lymph nodes secondary to the pulmonary metastasis, multiple pulmonary metastases, and pulmonary metastases requiring pneumonectomy.

4. A prolonged time interval between the resection of a primary lesion and the appearance of pulmonary metastases appears to be correlated with a favorable prognosis.

5. The removal of pulmonary metastases is safe, with little associated morbidity, and should be strongly advised when the proper criteria exist. At the present time, no other form of therapy offers a reasonable hope of survival.

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DISCUSSION

DR. KENNETH HARDY, Oakland, Calif.—We would like to present a case to reinforce what Dr. Wilkins had to say regarding osteogenic sarcoma.

[Slide] This 55-year-old woman had a left mid-thigh amputation in 1950 for osteogenic carcinoma of the tibia. She was perfectly well, did well on a prosthesis, and was very active until December of 1953 when we saw her because of recurring hemophysis of up to a half cupful of bright red blood per day for over a month. The film on the left reveals a mass in the left lower lobe, deep in the costophrenic sulcus. Diagnostic pneumoperitoneum and fluoroscopy revealed the mass to be in the lower lobe. On two separate occasions, bronchoscopy localized the bleeding as coming from the lower lobe bronchus.

With some apprehension we did a conservative resection, namely, a basilar segmental resection of the left lower lobe. The patient is now, 7½ years later, completely well. She was seen this last month.

DR. JOHN W. STRIEDER, Boston, Mass.—This is an important contribution to this interesting facet of pulmonary disease. As time goes on, we will operate upon more patients with more extensive and more unfavorable carcinomas in a much more radical way and, consequently, the problem of pulmonary metastases will confront us with increasing frequency.

An aggressive philosophy in dealing with neoplastic disease is supported by the knowledge, amply confirmed this morning, that single or even multiple pulmonary metastases are not necessarily prima facie evidence of widespread extrapulmonary dissemination of the disease.

It is important for us to emphasize this because there is still a natural reluctance on the part of the physician to subject his patients to an operation in a situation which has heretofore been considered without help or hope. Statistically, a patient with one cancer is more apt to develop a second primary lesion. Hence, radiographic demonstration of a shadow of increased density in the pulmonary field at any time after the apparently successful extirpation of a primary growth elsewhere may, in fact, indicate another primary tumor which may be a benign one.

Of 4 patients who had had cancers of the breast removed previously, 3 had benign tumors (two adenomas, one hamartoma) and one had a malignant tumor.

Our distribution in 29 patients who had 31 operations in a 15-year period, 1954 to 1961, was very similar to that obtained by Dr. Wilkins. [Slide] Our results from the point of view of survival are also very similar. We have 7, or 25 per cent, alive 5 to 15 years after the operation, and 11, or 40 per cent, survived 2 years. (In his series) there was one death due to renal shutdown because of a transfusion reaction.

DR. ADRIAN LAMBERT, New York, N. Y.—I want to compliment the authors for bringing to our attention the importance of a vigorous approach in the removal of secondary disease of the lung. It is interesting that in both the series which have been shown here so far, there was not one case in which the primary tumor originated in the lung.

One always should beware of the man who has only one case, but I do think it is appropriate to present a follow-up study on a patient who was brought to the attention of this Society in 1949. This was a 26-year-old girl who, in 1946 at the Roosevelt Hospital, had an adenocarcinoma of the left upper lobe removed by left upper lobectomy. One year later a metastasis in the right upper lobe was also removed by lobectomy. Six years after the removal of the primary tumor, when she was 4 months pregnant, a nodule was noted in her right breast, which proved to be a primary mammary carcinoma, for which radical mastectomy was performed. She subsequently delivered a normal boy.

The first slide shows the primary tumor, a 14 cm. solitary adenocarcinoma in the left upper lobe. She was symptom free when this mass was picked up on routine x-ray films. We removed this, along with eight nodes, all negative for tumor, although the pulmonary vein showed metastatic cells present within it [slide].

When a cough developed 11 months later, x-ray studies showed a metastasis in the right upper lobe [slide], and this was removed. X-ray examination a year ago revealed no evidence of recurrence. It is now 15 years after the removal of the primary tumor. The patient is living a healthy life, free of malignant disease. This case emphasizes the fact that primary carcinoma of the lung, although it has metastasized, can be arrested and, in a rare case, cured.

DR. CAMERON HAIGHT, Ann Arbor, Mich.—The paper by Dr. Wilkins and his co-authors is timely because sufficient opportunity has now been had to evaluate some of the late results of treatment of metastatic neoplasms. Dr. Wilkins has referred to the 6 cases which Dr. Alexander and I reported in 1947. Three of these patients have survived their metastatic disease and all of the 3 had metastatic spindle cell sarcomas. I believe it is of interest and also of encouragement to see the late results in these 3 cases.

The first patient in our series had had a neurogenic sarcoma removed from the forearm. She was seen one year later, in July, 1939, with a solitary lesion in the right lower lobe. A right lower lobectomy was done. She returned 7 months later with a large lesion in the left upper mediastinal region. Irradiation was advised but the patient refused to

accept the recommendation. She returned in June, 1940, at which time the lesion had shown additional and rapid growth. As an induced pneumothorax demonstrated the lesion to be within the lung, Dr. Alexander performed a left upper lobectomy in July, 1940. Even though the neoplasm was adherent to the superior mediastinum, it appears that the lesion was completely removed. The latest available roentgenogram of her chest in 1956 shows no evidence of recurrence. I have spoken with this patient recently and she has had no symptoms of recurrence. It is difficult to believe that such a result is possible but it demonstrates that a miracle can occur rarely in this condition.

The second of our patients to survive the metastatic disease is one who had had a spindle cell fibrosarcoma removed from the muscles of the thigh in December, 1940. He returned in September, 1942, with a small lesion in the upper lobe of the right lung. As biopsies of the thigh showed no evidence of recurrence of the sarcoma, a right upper lobe lobectomy was performed in the same month. The patient lived for 16¼ years at which time he died of an unrelated illness and without evidence of recurrence of the sarcoma.

Our third survivor had a spindle cell sarcoma of the popliteal region for which a high thigh amputation was done in March, 1943. He returned 2½ years later with a large lesion in the right lower lobe. A pedunculated extension of the lesion into the intermediate bronchus had resulted in atelectasis of the middle lobe. The middle and lower lobes were removed in September, 1945. The slide shows the pedunculated lesion extending above the level of transection of the intermediate bronchus. The patient is living without evidence of recurrence at the present time.

DR. OSLER ABBOTT, Atlanta, Ga.—I rise primarily to emphasize the frequency with which one may encounter endobronchial metastases which imitate bronchogenic carcinoma. We have had 13 cases of this syndrome. Studies show that 4 were primary gastric carcinomas, 3 were primary seminomas, 2 originated in the breast, 2 were fibrosarcomas, and there was one each of neurogenic sarcoma, Ewing's tumor, and hepatoma.

In 7 instances these metastases occurred 4 to 26 years after excision of the original tumor. In only 4 of these 13 patients was it considered advisable to attempt an excision of the metastasis. In all 4 instances, extensive lymph node involvement was encountered and no successes were achieved.

I wonder if Dr. Wilkins would comment on the frequency with which he has found lymph node involvement in cases of endobronchial metastases.

DR. ROBERT J. JENSIK, Chicago, Ill.—Our group at Presbyterian-St. Luke's Hospital and the University of Illinois have followed 82 patients of this type from 1941 to the present time. In this group 45 have been operated upon in 1956 or before. The five-year survivors number 10, or 22.2 per cent [slide].

Whereas others have achieved a very satisfactory result in the renal and adrenal group, our results have been rather dismal. In the series of utero-ovarian tumors, 4 of 12 patients operated upon have survived over 5 years.

In the miscellaneous sarcoma group, the patient who survived the longest (operated upon by Dr. Van Hazel in 1941) lived 20 years after removal of the metastasis and 32 years after amputation of the left lower extremity.

As indicated by Dr. Wilkins, a long period between recurrence and the primary operation results in a better outlook.

I would like finally to make a plea for accuracy in diagnosis, lest we interpret tumors as multiple primaries. Here [slide], as you will see, is a lesion in which squamous changes are seen which might make one think of a second primary pulmonary tumor. Actually, the lung tumor was the same as the breast tumor in which squamous cell features were pronounced. The lung tumor was a metastasis from the patient's breast carcinoma.

DR. WILKINS (Closing).—Dr. Gibbon, I would like to thank these six gentlemen who discussed the paper, and I want to answer Dr. Abbott's question about bronchial

metastases. We had a surprisingly low incidence in these 67 patients. There were only 3, but our experiences with these 3 were all bad. They all had positive lymph nodes and I presume that is the reason they did poorly.

We can sum up the discussion by making three points as the reason for an aggressive approach to this problem. The first is to establish a diagnosis. Unless we attack these lesions aggressively, we are going to miss second primary carcinomas of the lung and other lesions, as Dr. Strieder mentioned.

Second, we seem to get some good results when we least expect to get them.

Third, with few exceptions, we have no other reasonable approach to the problem.

BRONCHIOLAR CARCINOMA OF THE LUNG

A Review of 33 Patients

Hugh F. Fitzpatrick, M.D., Robert E. Miller, M.D. (by invitation), Malcolm S. Edgar, Jr., M.D. (by invitation), and Charles F. Begg, M.D. (by invitation), New York City, N. Y.

IN RECENT years we have been impressed by the apparent increased incidence of bronchiolar carcinoma of the lung at St. Luke's Hospital. Since 1953, more cases are occurring at our hospital; since then, 33 patients have been proved by strict histologic criteria to have this disease. Eleven cases were diagnosed in the past year alone.

This is not a local phenomenon. Before 1943 there were only a total of 25 cases reported in the literature.[1, 2] Since then, Storey and colleagues[3] reviewed 205 cases in 1953 and subsequently, to the best of our knowledge, 121 cases have been reported.[4-26] Whether bronchiolar carcinoma of the lung is, in fact, occurring more frequently or whether it is now being diagnosed more accurately is an interesting point for reflection. The important fact is that, today, bronchiolar carcinoma of the lung is becoming a less uncommon type of pulmonary malignancy. From our review of the literature, there are reported ten 5-year cures following surgery for this disease.[1-3, 27-29] This poor record undoubtedly reflects primarily a delay in diagnosis, which has allowed the disease to advance to a nonresectable stage. More reviews, therefore, of the clinical course of this disease are in order and a thorough search for reliable guideposts for diagnosis and therapy must be made.

No pulmonary malignancy is more interesting or more bizarre in its clinical course and yet less understood. Thirty-six different names have been proposed to describe this disease, which has been considered the most uncommon malignant epithelial tumor of the lung.[30] We have chosen the term *bronchiolar carcinoma* because, in careful review of the histopathology in these cases, the tumor cells occasionally are ciliated and often appear to spread into the alveolar spaces from the smaller bronchial radicles. The more recent literature supports this view.[8, 31]

MATERIAL

The records of the Pathology and Medical Records Departments of St. Luke's Hospital from Jan. 1, 1953, to Jan. 1, 1961, were reviewed and a total

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of 33 cases of bronchiolar carcinoma were found. Each case is fully documented histologically by surgical or autopsy specimen. Included are 17 males and 16 females. The mean age was 65.5 years, with a range of 37-82 years. Thirty patients were white, two were Negroes, and one was an Oriental.

SYMPTOMS

The chief complaint was stated in all but one case, but symptoms suggested pulmonary disease in only 14. Nineteen patients (58 per cent) were free of pulmonary symptoms except for a few with a mild chronic cough which had remained unchanged. Only 6 patients complained of cough as a major symptom. Four patients had chest pain, and one each had hoarseness and massive hemoptysis. Characteristically, the cough has been described as productive of voluminous amounts of frothy, watery sputum.[3, 7] However, in our series, the cough was either nonproductive or productive of a very small amount of nonspecific sputum.

The one patient presenting with hemoptysis, requiring whole blood replacement, also had a coincidental blood dyscrasia which may have led to a hemorrhagic diathesis. Only one other case of massive hemoptysis associated with this disease is recorded in the literature.[13]

Information with regard to smoking is recorded for 24 patients; 6 were nonsmokers, and the remaining 18 smoked an average of 20 cigarettes per day.

A history of pulmonary tuberculosis was given by 3 patients, but in each case this disease was considered arrested at the time of their admission to the hospital.

PHYSICAL FINDINGS

The physical examination was an unrewarding exercise in these patients. Twenty patients exhibited no thoracic physical findings; 13 had râles, dullness, or decreased breath sounds, but the location described correlated very poorly with the subsequently demonstrated pulmonary infiltrate on the roentgenogram. Certainly no pathognomonic pattern of percussion or auscultatory findings was observed. However, 4 patients (12 per cent) had clubbing. Cyanosis was not observed as an initial symptom.

DIAGNOSTIC STUDIES

The roentgenogram is the key to diagnosis in this disease. All but one patient had one or more abnormal preoperative or preautopsy roentgenograms. In 46 per cent of the patients the lesion was detected on a routine x-ray examination. Excluding those patients who were either in the terminal stages of their disease, or whose disease was an incidental finding (8 patients), 92 per cent had a single lobe lesion. In 84 per cent of this group, the solitary infiltrate was located in an upper lobe. Only 8 patients from our series are excluded from these statistics. None of these was a candidate for curative therapy—one had a microscopic lung tumor, while the remaining 7 had widespread disease when

first seen—since the diagnosis of bronchiolar carcinoma was made only after autopsy. Restated, then, 92 per cent of our patients in whom the diagnosis was made prior to death should have been candidates for resectional therapy at the time of their first abnormal roentgenogram of the chest.

Bronchoscopy is not considered helpful in the early diagnosis, since the lesion is more peripherally placed than the bronchogenic cancer. Thirteen of the 33 patients were subjected to bronchoscopic examination; in 3, gross findings consistent with malignancy were found; in 3, the findings were not recorded. All 13 patients had bronchial washings taken for Papanicolaou smears: 7 had Class I or II smears, one had Class III, 2 had Class IV, while information was not available about the remaining 3 patients.

Eight patients underwent appropriate scalene node biopsy. In 2, the lymph nodes contained carcinoma; 6 showed no malignancy.

Only 2 patients required thoracentesis for pleural effusion. One revealed a Class IV cytologic smear and the other Class I.

One patient had diffuse pulmonary disease with many different diagnostic opinions expressed after thorough study (J. H. Y., Clinical Group II). A lung biopsy, as described by Klassen,[32] showed bronchiolar carcinoma of the lung.

THERAPY

Twenty of the 33 patients underwent thoracotomy; 7 had nonresectable lesions and only biopsy was performed. The specimen obtained was diagnostic in each case. Thirteen were subjected to resectional therapy: 9 had lobectomy, of whom 3 had involvement of hilar nodes; 4 patients underwent pneumonectomy, with 3 showing spread to hilar nodes, and one free of nodal metastasis. The results of surgical therapy will be discussed further.

Cobalt^{60} irradiation was given to 6 patients—3 who had undergone a lobectomy, and 3 whose disease was diagnosed by open biopsy. All of these patients died, the average survival following diagnosis and the initiation of radiotherapy was 7.5 months. A similar group of patients whose disease was diagnosed by open biopsy (2 patients), pneumonectomy (4 patients), and lobectomy (2 patients) were not given postoperative irradiation. As in the first group, all of these patients died after an average postoperative survival of 7.7 months. Thus, this experience with these few patients indicates that radiotherapy is of limited therapeutic value in this disease. Two patients received 3,000 r, and 4 patients 4,000 to 6,000 r.

Several terminally ill patients in this series received nitrogen mustard in an attempt to palliate the disease. The numbers are too few and survival times too brief, but the clinical impression as recorded in the charts appears to indicate minimal response.

CLINICAL GROUPS

We arbitrarily have divided these patients into four groups, based on the length of time which elapsed from the initial abnormal pulmonary infiltrate

until the definitive diagnosis of bronchiolar carcinoma of the lung was made (Table I). Clinical Group I comprises 6 patients in whom there was a time lapse of 2 years or more; the average delay being 5¼ years, the maximum 12½ years. Two patients died after 4 and 3 years with repeatedly demonstrated pulmonary disease. They had undergone no attempted definitive diagnosis. Two patients had nonresectable disease at thoracotomy 2 and 12½ years after their lesions were first seen on x-ray examination; one has died, while the other is receiving terminal care. Another patient died 20 months after lobectomy which was performed 6 years after the first abnormal roentgenogram. The last patient developed neurologic complaints after 4 years with a known lung lesion; he is living with his disease following a craniotomy and excision of metastatic bronchiolar carcinoma.

TABLE I. CLASSIFICATION OF BRONCHIOLAR CARCINOMA OF THE LUNG: INTERVAL FROM ABNORMAL ROENTGENOGRAM TO DIAGNOSIS

	NO. OF PATIENTS	AVERAGE AGE AND RANGE (YEARS)	AVERAGE DELAY	RESULTS
GROUP I > 2 Years	6	62 (37-82)	5.25 Yr. (2-12½ Yr.)	2-No surgery; autopsy diagnosis 3-Nonresectable 2 living with disease 1 dead 1-Lobectomy; dead
GROUP II 6 Months to 2 years	2	54 (42-66)	14 Mo. (10-18 Mo.)	2-Operations 1 Lung biopsy; living with disease 1 Pneumonectomy; died postop.
GROUP III < 6 Months	17	64.7 (46-78)	5.2 Wk. (1 Wk.-4 Mo.)	17-Thoractomy 6 Nonresectable -- 35% 2 living with disease 4 dead 6 Resection; dead 35% 5 Post-lobectomy; alive and well ------ 30%
GROUP IV Unsuspected or incidental autopsy diagnosis	8	72.5 (47-82)	---	---

Clinical Group II includes 2 patients whose disease was diagnosed between 6 months and 2 years after the first abnormal x-ray findings. The average time lapse was 14 months. One patient died 4 days after pneumonectomy, which was performed 10 months after a pulmonary lesion was first detected. The surviving patient has bilateral disease documented by open lung biopsy (J. H. Y., Clinical Group II).

The third and largest group is comprised of 17 patients in whom 6 months or less elapsed between the first abnormal roentgenogram and definitive diagnosis. The average time lapse was 5.2 weeks. Two patients died following

scalene node biopsy and 2 died following open thoracotomy. Three more died following pneumonectomy and 3 after lobectomy. Two are receiving terminal care after their disease was diagnosed at thoracotomy. However, lobectomy was performed on 5 patients who were alive and well on April 1, 1961 (Table II).

TABLE II. PATIENTS SURVIVING LOBECTOMY WITHOUT CLINICAL OR X-RAY RECURRENCE

PATIENT	AGE	LOCATION OF LESION	SYMPTOMS	NODES	X-RAY THERAPY	FOLLOW-UP (MO.)
J. D.	61	LUL	Cough and hemoptysis	14/14 Positive	Yes	16
H. H.	62	LUL	None	Negative	No	23
A. L.	67	RUL	None	Negative	No	33
H. O.	78	LUL	Chest pain	Negative	No	37
H. M.	67	RUL	None	Negative	No	45

Clinical Group IV includes one patient whose tumor was an incidental autopsy finding. The actual cause of death was a resistant meningitis following craniotomy for glioblastoma multiforme. The bronchiolar carcinoma of the lung was found only on microscopic examination and therefore was an unsuspected finding. Seven patients, entering the hospital and receiving terminal care for disease undiagnosed until autopsy, are also included in this group. Of these 7, excluding one patient who survived 5 months in the hospital, the average length of hospital survival was 14.5 days. The average hospital stay for the entire group was 57.4 days.

CASE REPORTS

Clinical Group I.

A. K. (St. Luke's Hospital No. 30-16-67) was a 43-year-old white woman who was first admitted to the hospital in 1942 for gynecologic surgery. She gave a history of having had sputum positive for tuberculosis during previous months. X-ray examination (Fig. 1, *A*), showed a soft left upper lobe infiltrate. During an admission in 1946 for orthopedic complaints, a chest roentgenogram was reported as "negative," although the infiltrate was still present (Fig. 1, *B*). In 1950 she was re-admitted for thrombophlebitis, and a chest roentgenogram (Fig. 1, *C*) showed a "left upper lobe infiltrate, more prominent than on the film taken in 1946." She was discharged without further evaluation of her lesion. On admission in 1956 for evaluation of gastrointestinal complaints, a routine chest film demonstrated a definite increase in the left upper lobe mass (Fig. 1, *D*). She gave no history of smoking or cough. Her lungs were described on physical examination as clear. She underwent bronchoscopy, and both bronchial washings and biopsy of the left upper lobe bronchus failed to reveal evidence of malignant disease. On July, 24, 1956, she underwent a left upper lobectomy. Pathologic sections revealed bronchiolar carcinoma of the lung. Postoperatively, she developed acute cholecystitis and a cholecystectomy was performed. She did well for 16 months following operation, but died of metastatic disease 20 months after surgery. In the tabulations (see Table I) we considered this patient delayed 6 years from date of x-ray examination (Fig. 1, *C*) showing a definite increase in the size of the pulmonary lesion.

Clinical Group II.

J. H. Y. (St. Luke's Hospital No. 40-84-91) was a 66-year-old Chinese food handler who was subjected to chest x-ray studies in August, 1958, because of a cough. Confluent

right upper lobe infiltrates and diffuse nodular lesions throughout the remaining lung fields were seen. Repeated chest x-ray examinations showed no significant change until January, 1960, when marked progression of the right lung infiltrates was noted. A trial on INH (isonicotinic acid hydrazide) therapy from September, 1959, to January, 1960, produced no radiographic improvement. Repeated cultures for tubercle bacilli were negative. The patient was admitted for further diagnostic evaluation and on admission claimed a recent weight loss of 6 pounds. He smoked 3 cigarillos a day and had had a cough productive of small amounts of blood-tinged sputum for one year. On physical examination, 2+ clubbing was noted, and moist râles were heard at the base of the left lung and throughout the right lung. Chest roentgenogram on admission showed diffuse fibrosis of both lung fields, a dense

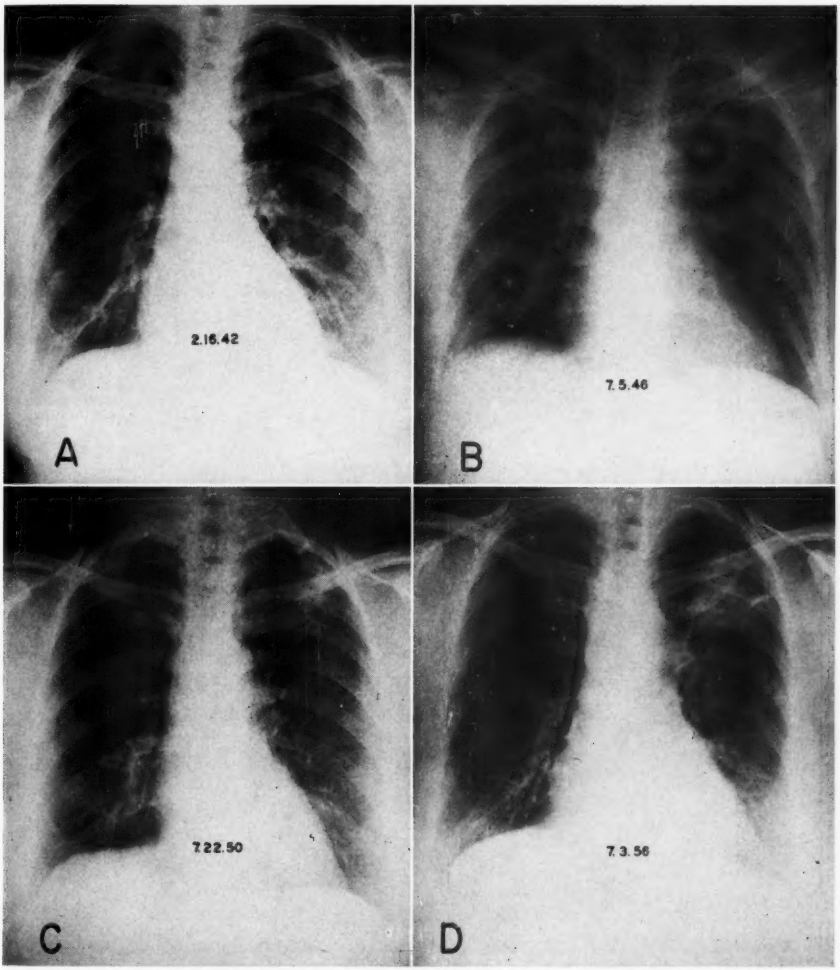

Fig. 1.—Serial roentgenograms of A. K. (Clinical Group I) showing progressive development of left upper lobe lesion. *A*, Lesion shown in left first intercostal space. *B*, Four years later lesion noted in same area, although x-ray examination was reported as "negative." *C*. Definite increase in size of lesion is noted (1950). *D* shows extensive involvement in identical area.

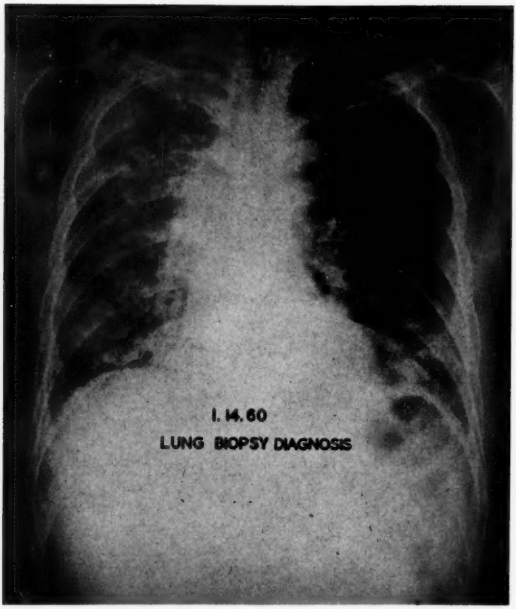

Fig. 2.—Preoperative chest roentgenogram of J. H. Y. (Clinical Group II) which demonstrates diffuse bilateral pulmonary disease, documented by right lung biopsy as bronchiolar carcinoma.

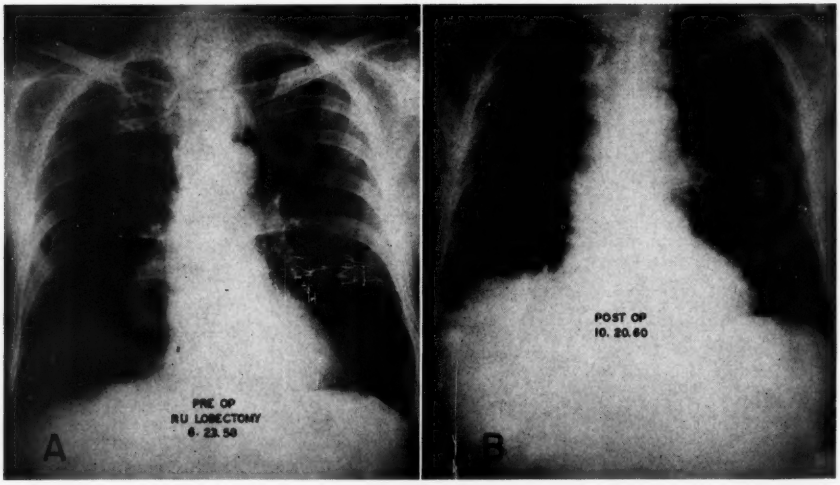

Fig. 3.—Chest roentgenograms of A. L. (Clinical Group III). *A*, Shows preoperative right upper lobe infiltrate. *B*, Chest film made 2 years and 4 months after operation, shows only postoperative changes.

right hilum, and a probable recent infiltrate in the right upper lobe (Fig. 2). On Jan. 18, 1960, a right scalene node biopsy showed chronic inflammation. An open lung biopsy on Feb. 1, 1960, revealed bronchiolar carcinoma of the lung. The patient was last seen in the clinic in March, 1961, complaining of a persisting cough associated with increased shortness of breath on exertion, but still able to earn a livelihood.

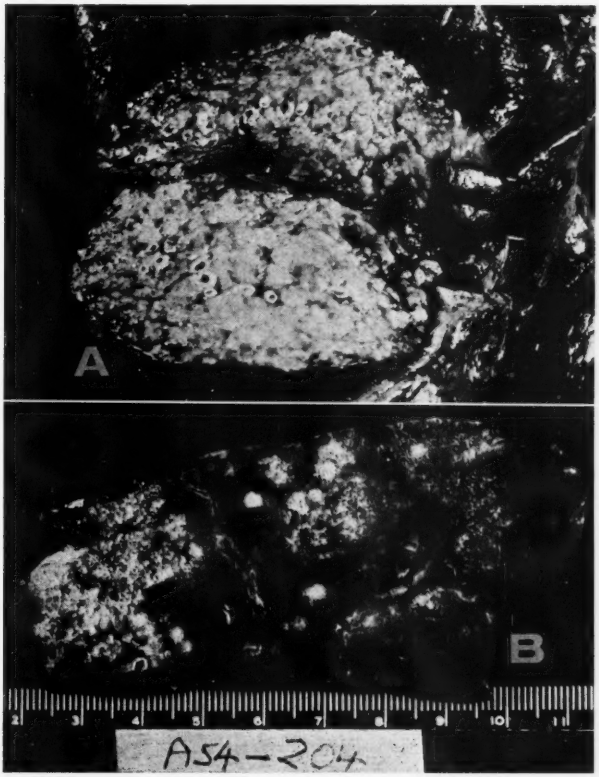

Fig. 4.—*A*, Pneumonectomy specimen: cross section of lung. Multiple tumor nodules are seen, many of which are confluent. Little normal parenchyma remains. *B*, Autopsy specimen. Multiple tumor nodules are scattered throughout the parenchyma, almost entirely replacing the upper lobe.

Clinical Group III.

A. L. (St. Luke's Hospital No. 36-28-69) was a 67-year-old white man who had a routine x-ray examination of the chest by his employer in April, 1958, which resulted in his admission to another hospital that month. Operation was advised on the basis of x-ray and bronchoscopic findings. The patient refused an operation and was referred to our clinic. He admitted to smoking one package of cigarettes a day and having a chronic nonproductive cough. Fine crepitant râles were heard in both lung bases posteriorly. No clubbing was noted. A chest roentgenogram demonstrated a 2 by 2.5 cm. lesion in the right upper lung field (Fig. 3). A right scalene node biopsy revealed no evidence of malignancy. On Aug. 11, 1958, a right upper lobectomy was performed and sections revealed bronchiolar carcinoma of the lung. Since discharge the patient has been closely followed by his private physician. A chest roentgenogram 19 months after thoracotomy showed only postoperative changes. As of April 1, 1961, 25 months after operation, he remains without complaints.

PATHOLOGY

There is considerable variety in the gross appearance of bronchiolar carcinoma. Some tumors appear as firm, tan, ill-defined areas not readily distinguished from an organized pneumonia. In other cases, the tumor is glistening, mucoid, and translucent. In either instance, the nodules may be single or multiple (Figs. 4, *A* and *B*). Microscopically, there is again great variation. The cells may be tall columnar, mucus-producing cells or irregularly shaped with abundant pink cytoplasm and dark, hyperchromatic nuclei. The well-differentiated cells have nuclei of normal appearance and such cells are occasionally ciliated (Fig. 5, *A*). They may be found lining spaces which have the configuration of the original pattern of the lung (Fig. 5, *B*); in the best differentiated areas, these cells form only a single layer. More commonly, papillae

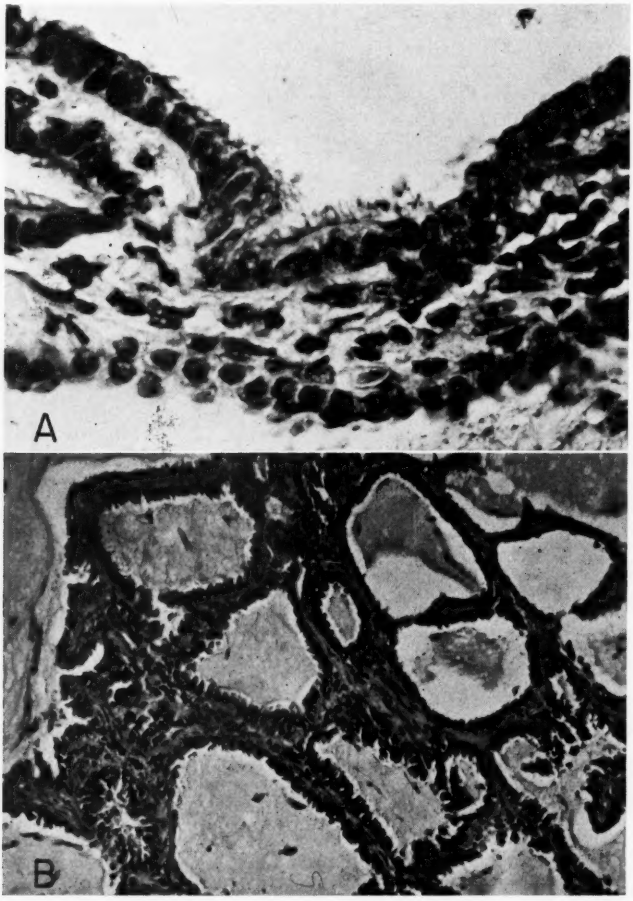

Fig. 5.—*A*, Ciliated neoplastic cells. (Orig. mag. ×360.) *B*, Cuboidal and columnar cells line the alveolar spaces and for mucin which fills the lumina. Pathologic Grade I. (Orig. mag. ×125.)

grow into the lumen of these spaces (Fig. 6, *A*). The less differentiated tumors have irregular acinar structures in between the larger spaces (Fig. 6, *B*); and in poorly differentiated tumors, only single cells and small clusters of cells can be found, with no evidence of alveolar space or acinar formation (Fig. 7, *A*).

These cellular characteristics form a convenient basis for dividing these tumors into four groups, according to the degree of histologic malignancy. Tumors with tall columnar, mucus-producing and, perhaps, ciliated cells lining larger spaces in a single layer are obviously the most differentiated—these are designated as Grade I. Poorly differentiated tumors composed of small eosinophilic cells, with hyperchromatic nuclei forming small and infrequent spaces, are designated as Grade IV. Assigning Grade II or Grade III is more difficult

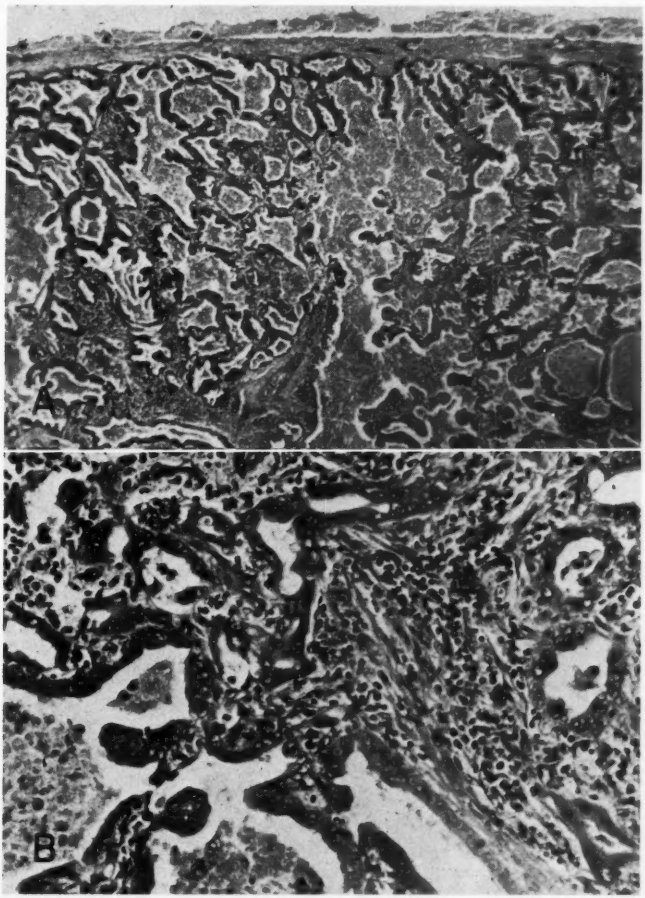

Fig. 6.—*A*, The alveolar pattern is noted. Cuboidal and columnar cells line the spaces and form papillae. Pathologic Grade II. (Orig. mag. ×40.) *B*, Irregular acinar structures are seen extending into the stroma. Pathologic Grade III.

but is based on the degree of cellular anaplasia and failure to form well-defined structural units. Multinucleated tumor cells may be found in Grades II, III, and IV (Fig. 7, *A*).

A systematic attempt was made to place each of the tumors from the patients into these four groups, and then to relate the microscopic appearance to the clinical behavior of the particular patient. It was soon apparent that this theoretical correlation did not exist. In many cases, the pathologic grading varied in different areas of the same tumor. In other cases, a resected tumor would not have the same pathologic grade as tumor in metastatic sites. Moreover, the length of time a patient harbored his tumor, the extent of local or

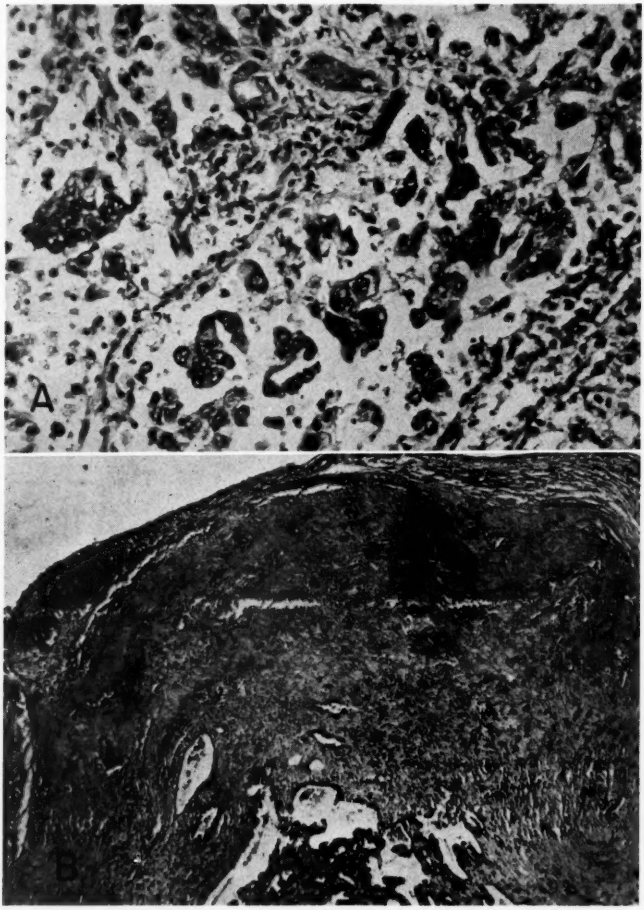

Fig. 7.—*A*, An area of poorly differentiated bronchiolar carcinoma containing many neoplastic giant cells. Pathologic Grade IV. (Orig. mag. ×150.) *B*, An old hyalinized scar seen at the lung periphery. Spaces lined by neoplastic cells are noted in lower portion of field. (Orig. mag. ×30.)

metastatic involvement, the amount of symptomatic debility—none of these things could be correlated with the estimate of histologic malignancy. This was found to be so, even when each tumor was grouped according to the most anaplastic area found. Our survivors include patients who had tumors resected which were classified as Grade II through Grade IV.

There was an impressive association of fibrosis with these tumors, a prominent feature in 67 per cent of these cases. In some instances, there is clearly an old hyalinized scar immediately associated with the tumor (Fig. 7, *B*), giving the impression of having antedated the formation of the tumor. In two instances this scar has the configuration of an old infarct, and recanalized vessels are seen at the periphery. In one instance, fibrocaseous tuberculosis is present in and about the tumor. In other cases, alveolar outlines are still recognizable in some places and the alveolar spaces are filled with maturing fibrous tissue; low columnar or cuboidal cells sometimes form a single layer along the alveolar membrane. These areas merge with areas of definite tumor. In the majority of instances, however, it is not possible to state whether the fibrosis preceded the formation of tumor or represents a host reaction to the growth of the tumor.

DISCUSSION

Early surgery is the best hope for cure of this disease. An appalling delay has occurred in establishing even the diagnosis, and then treatment, in many of these patients with a clearly demonstrated but unexplained abnormal lung lesion. This delay has been a lethal factor, as demonstrated by patients in Clinical Groups I and II. An unresolved pulmonary infiltrate is not an innocent lesion—it may be cancer.

Five patients, 30 per cent of those operated on within 6 months following the initial abnormal chest roentgenogram (Clinical Group III), are alive and free of disease 16 to 45 months after operation. Even earlier surgery in this group of patients undoubtedly would have improved the resectability and possibly the cure rate.

Lung "scars," persistent "nonspecific" radiographic lung infiltrates, or unresolved pneumonia should not be regarded as benign and simply ignored. Very careful and frequent follow-up examinations should be made on patients with such lesions. If the lesion is expanding and it remains histologically or etiologically unidentified, it is a danger sign. It may be a lung cancer or a lesion which may predispose to bronchiolar carcinoma of the lung. Surgery, to permit diagnosis and to eradicate the disease, must be a foremost consideration in such patients.

The importance of evaluating undiagnosed x-ray lesions is stressed by a close observation of the data obtained from the arbitrary division of the patients into four clinical groups. Patients categorized in the fourth clinical group are automatically precluded from the possibility of curative therapy. There remain 25 patients, 20 of whom underwent thoracotomy in the hope of ridding them of what most assuredly is a fatal disease unless completely excised. In Clinical Group I, only one patient (17 per cent) was subjected to resectional

therapy and this patient succumbed to her disease shortly thereafter. Despite the fact that 4 of these 6 patients (67 per cent) were initially asymptomatic, but showed a pulmonary lesion on routine x-ray examination of the chest, all died following a fruitless delay in the hope that this silent, but unexplained, infiltrate would by itself disappear. In Clinical Group II, one of 2 patients had resection performed, but died shortly after operation. In Clinical Group III are included the greatest number of patients of the series. None was considered to have been delayed unduly in proper management, since complete diagnostic and, in some cases, therapeutic measures were initiated promptly after the abnormal chest roentgenogram was seen. It is true that even in this "favorable" group, one does not know the duration of the disease prior to its detection by x-ray examination. Still, the most objective appraisal of the surgical approach to this disease must be judged from the results of this group. Included are 17 patients, only 6 of whom had symptoms suggestive of primary pulmonary disease. Sixty-five per cent of this group were being evaluated for a lung lesion found on routine x-ray examination of the chest (9 patients), or an incidental finding on a roentgenogram taken for deltoid bursitis (1 patient) or rib trauma (1 patient).

The unquestionable theoretical advantage prompt therapy affords these patients was only partially realized. Six of them, or 35 per cent of the group, had widespread intrathoracic disease at thoracotomy or scalene node biopsy. Six additional patients (35 per cent) had resection but have since succumbed to their disease. Of the remaining 5 patients, all are alive with no sign of recurrent disease 16 months, 23 months, 33 months, 37 months, and 45 months, respectively, after operation. All of these patients had lobectomies. (None of the post-pneumonectomy patients have been cured.) Thus, 30 per cent of those patients whose demonstrated lung infiltrate was aggressively approached surgically have no sign of recurrent disease now—16 to 45 months postoperatively. This in no way predicts or assures a permanent cure in these patients, but it is a promising step in that direction.

Eighteen per cent of our patients (Clinical Group I) were followed 2 years or more with "progress" x-ray studies for unresolved radiographic findings. Interestingly enough, Rigler,[14] in reviewing roentgenograms of all lung cancers, noted that in all but 2 of the 100 cases there was evidence of an abnormality (by x-ray) in the area of the lung later proved to be the location of the carcinoma. Furthermore, he noted that these x-ray findings had been present in almost all cases long before any significant symptoms were apparent. Fifty per cent showed positive x-ray findings more than 2 years prior to either the development of symptoms or definitive diagnosis. One of his cases was of a bronchiolar carcinoma of the lung. Arany[10] concludes, with reference to bronchiolar carcinoma of the lung, that the lesion may remain asymptomatic for a considerable period of time. He stresses that a solitary pulmonary peripheral nodule, although asymptomatic and showing no changes in size for a long period of time, may be malignant. Overholt[20] notes that 20 per cent of his 15 patients with operable bronchiolar carcinoma had positive x-ray findings 2 to

3 years before surgery, and one case for 8 years; yet all these patients had nodes free of metastases. Others have presented one or 2 cases with delays of 4 to 6 years between x-ray examination and symptoms and operation.

Auerbach and colleagues[33] found an increasing incidence of pulmonary fibrosis secondary to pneumonia. In 1930 the incidence was 5 per cent; in 1940, 7 per cent; and 12 per cent of 307 autopsies between 1946 and 1950. It was their opinion that this increased incidence was due either to a higher incidence of viral pneumonia, or to a failure of inflammatory resolution in bacterial pneumonia. They postulated further that antibiotics may retard the host response to bacterial infection by depressing leukocyte infiltration into the involved area, which, in turn, prevents an adequate enzymatic reaction from the polymorphonuclear cells to cause resolution of the inflammatory or fibrinous exudate. Organization and fibrosis thus occurs.

In bronchiolar carcinoma of the lung there is an even more impressive incidence of associated fibrosis and chronic inflammation—67 per cent in this series. These findings agree with those of Beaver[18] and Spain,[12] both of whom believe that chronic inflammation predisposes to this disease. The apparent predilection to upper lobe involvement, as also occurs in pulmonary tuberculosis, and the increasing incidence of this lesion (reported in the literature since the antibiotic era) do suggest more than a chance relationship to pulmonary infection.

SUMMARY

From the review of these 33 patients we consider it pertinent to stress:

1. Bronchiolar carcinoma of the lung is increasing in incidence and should no longer be considered a rare disease.

2. Approximately 60 per cent of the patients with this disease are asymptomatic. The disease is predominantly a solitary lesion, usually in an upper lobe.

3. An appalling delay has occurred in establishing the diagnosis and surgical treatment in many of these patients who have died or who are receiving terminal care with widespread disease.

4. Bronchiolar carcinoma of the lung is clinically and pathologically a bizarre and unpredictable lesion. There is an impressive association of inflammation and fibrosis with this disease.

5. X-ray examination of the chest is the key to diagnosis in this disease. Persistent nonspecific lung infiltrates or unresolved pneumonias, clearly demonstrated in x-ray studies, are danger signs: they may be pulmonary cancer, or a lesion which predisposes to bronchiolar carcinoma of the lung.

6. Today, bronchiolar carcinoma of the lung is rarely, and should never be allowed to become, a diffuse bilateral disease afflicting a coughing, cachetic, terminally ill patient. Only by early recognition and prompt surgical intervention can the dismal progress made thus far in treating this disease be improved.

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Discussion

DR. JOHN E. CONNOLLY, San Francisco, Calif.—In general, we are in agreement with what has been said about bronchiolar cell carcinoma. However, we were in a quandary about these tumors until we consulted our surgical pathologist, Dr. Stanton Eversole, and some of his observations have interested us a great deal and we thought that you might like to hear them.

[Slide] This is the appearance of what we like to call the typical picture of alveolar cell carcinoma. You will notice that there are tall columnar cells, which are mucus-producing, and mucus-secreting. There lesions may be diffuse or localized. They often give symptoms and they have a very poor prognosis.

In the more benign type of this particular lesion, we like to use the term "pulmonary adenomatosis."

[Slide] This is a lesion that is often seen as an incidental finding at postmortem. We consider this to be a circumscribed adenomatous malformation, benign in nature, and you will notice that the cells are cuboidal, nonciliated, and do not produce mucus.

[Slide] This is a similar gross appearing lesion. However, when you examine the cuboidal cells in this specimen, there are definite malignant characteristics and this is a lesion we like to call "bronchiolar cell carcinoma."

We have collected 9 cases of this lesion in the San Francisco Bay area. Dr. Boyd, working in our department, has done a very thorough search for these cases. All of these lesions were found incidentally on routine chest films and were resected. The follow-up study has covered 9 months to 5 years and all patients are living and well.

We feel, then, that there is a lesion which we like to call "bronchiolar cell carcinoma" as opposed to "alveolar cell carcinoma," in which the prognosis is excellent, and I think that if your surgical pathologist examines the microscopic picture carefully, and finds the picture that we call "bronchiolar cell carcinoma," he can predict an excellent prognosis. I would be very interested to examine the slides on the cases that Dr. Fitzpatrick has presented as I think we can predict the patient's prognosis from the microscopic appearance of the tumor.

DR. JOHN W. BELL, Seattle, Wash.—I would like to add a word of caution about the acceptance of lobectomy as necessarily the correct operation for a localized bronchiolar carcinoma.

[Slide] We have recently reviewed our experience with 21 cases of bronchiolar-alveolar carcinoma with a minimum follow-up of 3 years.

Twelve patients were treated with resectional surgery, approximately half with pneumonectomy and half with lobectomy, and, of the 12, only 3 are alive at the present time—1 over 3 years after lobectomy, 1 over 3 years after pneumonectomy, and another alive over 9 years after pneumonectomy. The mean survival time of the group is 27 months.

[Slide] I would like to emphasize one important point about the pathology of this disease and the way in which it may relate to the surgical treatment—this is the great propensity for bronchiolar-alveolar cell carcinoma to invade the intrapulmonic lymphatics (in over half the cases in our series), as well as the fact that tumor cells are found in the alveolar spaces in all of our cases.

[Slide] This is an example of tumor cells lying free in alveolar spaces in the lower lobe of a patient who had a pneumonectomy for a left upper lobe lesion.

[Slide] This shows the way in which free tumor cells attach themselves to the alveolar wall and you see the invasion of the fibroblasts fixing the tumor cells at this point.

Therefore, we feel that pneumonectomy may be the treatment of choice in bronchiolar-alveolar carcinoma because of the possibility of intrabronchial aerogenous spread from the upper to the lower lobe.

DR. PHILIP M. IKINS, Syracuse, N. Y.—Dr. Bugden, Dr. Staley, and I have 17 cases of bronchiolar-alveolar cell carcinoma which we have followed for the past 10 years. In these cases, we have a survival rate of around 80 per cent.

They were all upper lobe lesions, they were all peripheral, and they were all diagnosed by x-ray studies, but at the time of x-ray examination these small peripheral lesions had one symptom which was common to most of them—a cough with hemoptysis.

We have made preoperative diagnosis after the first 5 cases, within a range of about 50 per cent accuracy, of bronchiolar-alveolar cell carcinoma with a cough, slight to massive hemoptysis, and a small peripheral carcinoma of the upper lobe.

DR. FITZPATRICK (Closing).—I would like to thank the discussers for their comments and to add a few brief words about the discussion. I believe, Dr. Connolly, we have no great difference on the histology. I didn't have time to show several slides, but the 5 patients who are still living, based on the grading which really doesn't mean too much, vary from Grade I to Grade IV. The histology is extremely variable in any one given tumor. There may be areas of papillation into the alveolar spaces; there may be associated old hyalinized scar; or there may be no acinar or alveolar structures left but simply replacement by multinucleated giant cells. This, of course, is in the most anaplastic type.

Concerning pneumonectomy, we have had no survivors among patients who had a pneumonectomy. I am skeptical about pneumonectomy as a means to prevent spread of the tumor into an adjacent lobe—as the same possibility would still exist that it may spread to the opposite lung. I am not so sure that pneumonectomy is necessarily the answer.

Our findings do not agree with those of the last discusser who reports an 80 per cent cure.

I would like to conclude by saying that we are in no way assured that the 5 survivors from 16 to 45 months will become 5 year survivors. We remain hopeful, however.

ROUTINE USE OF THE CARLENS DOUBLE-LUMEN ENDOBRONCHIAL CATHETER

An Experimental and Clinical Study

Robert W. Newman, M.D., George E. Finer, M.D. (by invitation), and James E. Downs, M.D. (by invitation), Knoxville, Tenn.

In 1949 Carlens[1] introduced a double-lumen endobronchial catheter for bronchospirometry. Since that time the subject of the use of the Carlens catheter in ventilatory function has been thoroughly explored and elucidated by Gaensler[2] and Gaensler, Maloney, and Björk.[3] The Carlens catheter was first used for anesthesia in thoracic surgery by Björk and Carlens[4] in 1950 for the prevention of spread of secretions from one lung to the other during pulmonary resection. Occasional reports have appeared in the literature in the past decade describing experiences with the use of the Carlens catheter for anesthesia in intrapleural procedures.[5, 6] Björk, Carlens, and Friberg[5] in 1952 reported on the use of the catheter in 500 cases and advocated that the method receive wider application in pulmonary surgery. No additional enthusiastic reports appeared until 1958 when Jenkins and Clarke[7] advocated the routine use of the Carlens catheter in pulmonary surgery. There are now over 1,130 cases reported in the literature encompassing all types of thoracic surgery in which the Carlens catheter has been employed.[5-9]

The generally accepted advantages may be listed as follows: (1) the catheter will prevent a patient from drowning in his own secretions; (2) it will make possible repairs of the bronchus or trachea; (3) it may prevent spread of the patient's disease to the contralateral lung; and (4) it will permit the removal of secretions while still providing the patient with an effective tidal volume.

Initial objections to the use of the Carlens endobronchial catheter for anesthesia arose from the resistance to the flow of gas through such a tube. The resistance figures are available and they are not prohibitive.[10] Another major objection which has prevented wider application is the difficulty in the introduction and placement of the catheter. By using the method as described by Lynch and Feraru,[11] we have found this objection to be no problem. A few isolated instances of accidents have been reported with regard to placement of the catheter, but it has been well established on the basis of clinical trial that this is a relatively safe method.

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METHODS AND CLINICAL EXPERIENCE

For the past 24 months we have used the Carlens endobronchial catheter in 200 consecutive patients. The age of these patients has ranged from 14 to 76 years. There were 120 males and 80 females in this group. The indications for operation are listed in Table I.

TABLE I. CONDITION FOR WHICH OPERATION WAS PERFORMED

DISEASE	NO. OF CASES
Tuberculosis	96
Carcinoma	39
Bronchiectasis	34
Pleural disease	13
Lung abscess	5
Miscellaneous	13
Total	200

The operations performed on this group of patients are presented in Table II.

TABLE II. OPERATIONS IN WHICH CARLENS CATHETER HAS BEEN USED

OPERATION	NO. OF CASES
Pneumonectomy	23
Lobectomy	58
Bilobectomy	13
Lobectomy and segmental	23
Segmental	44
Decortication	8
Thoracoplasty	6
Thoracotomy	12
Bronchopleural fistula	6
Miscellaneous	7
Total	200

TECHNIQUE OF INTUBATION AND MAINTENANCE OF ANESTHESIA

During the course of this work, we have found many details of technique that serve to safeguard the patient and should be emphasized. The choice of anesthetic agents used is secondary in importance to the methods. Anesthesia is induced with an ultra-short acting barbiturate; then succinylcholine by constant drip is added to produce apnea. As soon as this is achieved, the patient is hyperventilated with 100 per cent oxygen for several minutes;[12] the ventilation is interrupted to spray the larynx and trachea with a topical anesthetic. The Carlens catheter is then introduced as described by Lynch.[11] The left bronchial tube is positioned anteriorly and introduced into the glottis. The catheter is then rotated 180 degrees counterclockwise so that the carinal hook now faces anteriorly at which point the hook passes easily through the glottis. The catheter is finally rotated clockwise 90 degrees and advanced until the hook engages the carina and then it is fixed in place. The normal curve of the tube is such that the left bronchial extension pushes the catheter to the right side of

the trachea and ensures that the hook will be caught at the carina. With the catheter connected to the gas machine by means of a double adapter, the right lung connector is occluded while the distal (left bronchial) cuff is inflated until there is no leak. The right bronchial tube is then opened and the proximal (tracheal) cuff is inflated so that the whole circuit is air tight. Finally, the correct placement is checked by auscultation over each lung, first with both lungs aerating and then with each occluded in turn. With practice, all of this can be done within a short period of time so that the previously well-oxygenated patient shows no change in the pulse rate.

An important consideration is that the largest possible catheter be used in order to minimize resistance and turbulent air flow. The Carlens catheter is available in sizes 35, 37, 39, and 41 Fr. In Table III is shown the tabulation of the size catheters used in our 200 patients. In the past the catheters used have been smaller than need be. We have found that most adult males can be intubated, if properly relaxed, with a 41 Fr. catheter and most adult females with a 39 Fr. A 35 Fr. is the smallest size and this is rarely used. Since, under conditions of turbulent air flow, the resistance varies inversely with the fifth power of the radius of the catheter this increase in size is most important.[10] The large-size Carlens catheter allows streamline flow at all flow rates of dry air.

TABLE III. CARLENS CATHETER SIZE USED

SIZE	NO. CASES
35 Fr.	9
37 Fr.	66
39 Fr.	60
41 Fr.	65
Total	200

Heretofore, the use of the Carlens tube has been considered dangerous by some because adequate ventilation was difficult to maintain. To ensure adequate ventilation, a mechanical ventilator is used, preferably of the pressure-limited type with the pressure changed to meet changing conditions and yet give the desired volume. The remainder of the anesthesia may be conducted with a semi-closed system utilizing the inhalation agent of choice; succinylcholine is added in amounts sufficient to keep the patient apneic and preclude coughing when the hilus is manipulated. Under these conditions there has been no difficulty in producing minute volumes of 10 to 15 L. per minute while using one lung and positive pressures under 35 cm. of water.

The lateral position has been used routinely in these patients. The patient is ventilated on both lungs until the pleural cavity is opened. Thereafter ventilation of the operated lung is tailored to expedite the surgery. The catheter to the operated lung is occluded completely when maximum exposure and minimum motion is required; or it may be partially occluded by a thumb-screw to facilitate segmental stripping. In many cases the lung has been kept completely atelectatic

for as long as 2 hours with no gross changes in blood pressure or pulse rate. There has been no difficulty in re-expanding the atelectatic lung after prolonged collapse, providing that secretions are removed first. Secretions from the operated side can be removed as soon as detected throughout the operation, since ventilation of the patient need not be interrupted. The location of secretions on the right or the left is easily identified and removed via a plastic catheter suction.

In operations for left pneumonectomy it is necessary to withdraw the left bronchial catheter into the trachea at the time the bronchus is to be transected. The patient then is maintained on endotracheal anesthesia until the operation is completed. A right endobronchial catheter recently described by White[13] would eliminate this problem.

ALTERED PHYSIOLOGY

The physiologic problems involved in the temporary venoarterial shunt resulting from acute atelectasis of the operated lung have been studied. Björk, Carlens and Friberg,[5] in 1953, studied 3 patients with arterial blood gas studies during acute atelectasis. When one normal lung is suddenly deflated, the same amount of blood passes through the nonventilated lung without being oxygenated. Gibbon[14] recently has measured the volume of blood flow in acute atelectasis in dogs and found it to be normal for the first hour and then to decrease gradually. However, in diseased lungs being operated upon, the pulmonary artery blood flow has already been decreased in the involved lung; in addition it has been shown by Rothstein, Landis and Narodick[15] that, in the lateral position for bronchospirometry with an intact, closed chest, there is increased oxygen absorption by the down lung. It is suggested that the cause of this increase is the effect of preferential gravity flow in the pulmonary artery to the down lung. This phenomenon probably is enhanced in the open thorax and in prolonged, acute atelectasis over 1 to 2 hours.

The physiologic effect of this temporary venoarterial shunt was measured in 6 mongrel dogs. The dogs were anesthetized and right thoracotomy was performed. Arterial blood oxygen, carbon dioxide, and pH values were obtained (1) preoperatively, (2) after occlusion of the right bronchus for 30 minutes, (3) after occlusion of the right pulmonary artery and the right bronchus for 30 minutes, and (4), finally, after return to a normal state with open thorax for 30 minutes. The results as pertains to oxygen saturation are shown in Fig. 1. There was a mean decrease of 10 per cent in arterial saturation due to the venoarterial shunt but, when eliminated by occlusion of the right pulmonary artery, there was a mean rise of 6 per cent in the arterial oxygen saturation. The arterial carbon dioxide content and arterial pH values remained constant with only minor fluctuations during the experiments.

The effect on arterial blood pH is shown in Table IV. A group of 5 patients in whom an endotracheal catheter was used for ventilation is compared with a group of 9 patients in whom a Carlens endobronchial catheter was used. The

preoperative arterial blood pH determinations were from brachial or femoral artery samples with the patient sedated but breathing room air. The postoperative samples were taken from the aorta at the completion of the resection but with the chest open and the operated lung still atelectatic in the Carlens catheter group. The two groups compare favorably as far as the pH determinations are concerned.

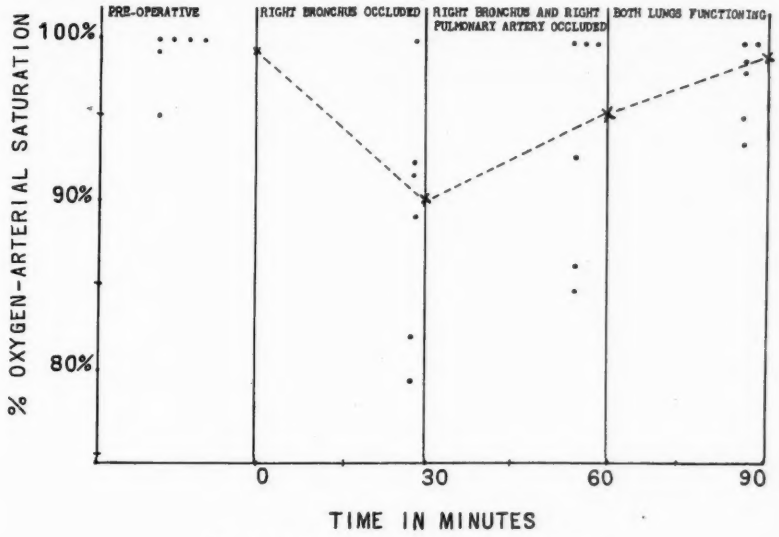

Fig. 1.—Animal experiments—6 mongrel dogs.

TABLE IV. ARTERIAL pH—(38°)

ENDOTRACHEAL CATHETER			CARLENS CATHETER		
PATIENT	PREOP.	POSTOP.	PATIENT	PREOP.	POSTOP.
S. F.	7.51	7.63	H. S.	7.44	7.31
V. L.	7.49	7.31	F. T.	7.51	7.51
J. L.	7.42	7.41	R. T.	7.47	7.43
R. L. H.	7.32	7.36	P. C.	7.33	7.45
V. W.	7.35	7.44	Z. D.	7.45	7.38
			E. D.	7.47	7.38
			G. H.	7.45	7.45
			B. M.	7.39	7.48
			S. R.	7.52	7.34

A group of 4 patients undergoing resectional surgery for tuberculosis with Carlens catheter intubation and using the acute atelectasis technique, as described above, are compared in Fig. 2. The preoperative arterial oxygen determination was made with the patient sedated and breathing room air; the second arterial oxygen determination was made at the conclusion of the resection with the operated lung still atelectatic. The final arterial oxygen determination was made with the patient extubated and breathing room air at the completion of the closure of the thoracotomy wound. The operated lung in this group was

atelectatic after the pleura was opened from 1½ hours up to 2½ hours. The arterial oxygen saturation is seen to be satisfactory in 3 of the 4 patients. In the poor-risk patient with only 85 per cent saturation at the end of the resection, there was a marked rise over the preoperative arterial oxygen saturation level.

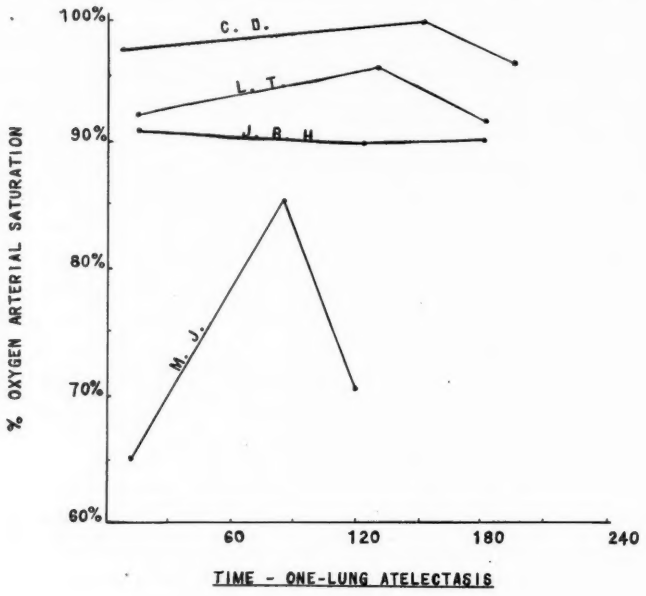

Fig. 2.

COMPARATIVE DATA OF ENDOTRACHEAL ANESTHESIA AND CARLENS ENDOBRONCHIAL ANESTHESIA

In Table V are presented comparative data obtained from 2 patients, each having had a right upper lobectomy for tuberculosis and presenting essentially the same preoperative ventilatory function. The only difference in the surgical management was that one was ventilated during anesthesia by means of an

TABLE V. ARTERIAL BLOOD DATA

		pH (38°)	pCO_2	CO_2 CONTENT	% O_2 SATURATION
Endotracheal Catheter					
V. W., right upper lobectomy	Preoperative	7.35	28.5 mm. Hg	31 Vol. %	100
MBC—35 L./min. (41%) VC—2.1/L. (61%)	Postoperative	7.44	17.4 mm. Hg	26.5 Vol. %	92
Carlens Catheter					
C. D., right upper lobectomy	Preoperative	7.48	25 mm. Hg	42.6 Vol. %	100
MBC—60 L./min. (62%) VC—3.0/L. (75%)	Postoperative	7.38	36 mm. Hg	41.5 Vol. %	97.7

endotracheal catheter, whereas in the other patient a Carlens catheter was used and the operative lung remained acutely atelectatic during the resection. The data compare favorably; both patients made an uneventful recovery.

In Fig. 3 are depicted the arterial oxygen saturation data on a poor-risk patient undergoing a preresection thoracoplasty using an endotracheal catheter, and operated upon in the lateral position, compared with the data obtained on the same patient using the Carlens catheter during a right upper lobectomy for cavitary tuberculosis and with one lung atelectatic for 1½ hours. The arterial saturation fell from 84 to 78 per cent during this period of atelectasis, and immediately postoperative with spontaneous breathing and chest closed, a value of 70 per cent arterial oxygen saturation was obtained. This patient died on the ninth postoperative day from ventilatory insufficiency with resultant ventricular fibrillation.

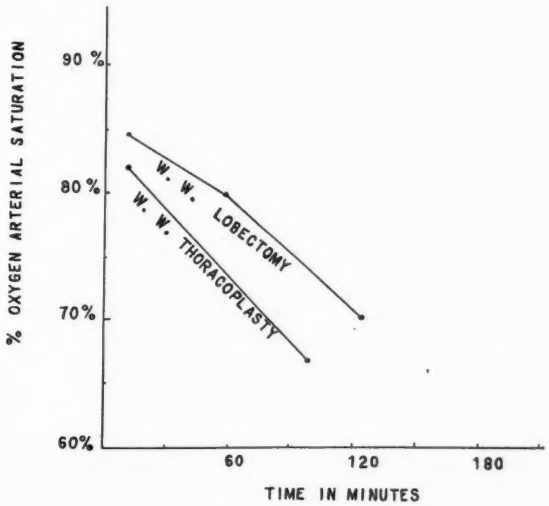

Fig. 3.

DISCUSSION

There are numerous definite advantages to the routine use of the Carlens endobronchial catheter for thoracic anesthesia. These may be divided into anesthesia advantages and surgical advantages.

Anesthesia Advantages of Carlens Catheter.—(1) Secretions are confined to the operative side. (2) Improved ventilation in bronchopleural fistula. (3) Tracheal or bronchial repair facilitated. (4) Adequate ventilation possible while aspirating the catheter and bronchus.

Surgical Advantages.—(1) There is wider exposure. (2) Hilar dissection is made easier. (3) Retraction of lung is not necessary. (4) Open technique of bronchus closure made possible. (5) Bronchoplastic techniques made easier. (6) There is less theoretical danger of cancer cell emboli.

These advantages lead to a more satisfactory technical procedure for both the surgeon and the anesthesiologist and result in a better operation for the patient. Indeed, some of the extremely difficult and sometimes impossible procedures are made possible and the routine resections are made easier. The lung can be deflated and left atelectatic while hilar dissection and ligation is performed. The bronchus may be divided and left open at any stage during the operation. A proximal bronchial clamp is never necessary. Bronchoplastic procedures are made technically easier. We have measured the tidal volume with one lung anesthesia on a Monaghan flowmeter interposed in the circuit and find that more effective ventilation is possible with this technique than when strong retraction is used to expose the hilar structures if both lungs are being ventilated.

Fig. 4.

There are only two absolute contraindications to the use of the Carlens catheter: (1) a glottis or trachea too small to admit the 35 Fr. catheter, and/or (2) a stenosis of the left main bronchus. White[13] described a similarly designed catheter for right endobronchial intubation which has a slot in the right bronchial cuff to permit ventilation of the right upper lobe; the left lung is thus ventilated via the tracheal cuff. We have had no experience with this catheter.

Preoperative bronchoscopy and bronchograms will reveal the patient in whom difficulty with intubation can be anticipated (Fig. 4). A bronchogram

showing horizontal displacement of the left main bronchus with resultant mechanical distortion of the left lower lobe bronchus would make positioning of the Carlens catheter difficult if not impossible.

COMPLICATIONS AND RESULTS

In our series of 200 patients we have had 15 complications ascribed to the catheter (Table VI).

TABLE VI. CARLENS CATHETER COMPLICATIONS

Cardiac arrest; death	1
Malposition	6
Failure to intubate	1
Anoxemia, one lung atelectatic	2
Suction catheter into pleural space via bronchial stump	2
Laryngitis, traumatic	3
Total	15

All of these complications were relatively minor with one exception; a death, early in the series, which probably was related to either malposition or inappropriate use of the catheter. This patient was an emphysematous, coal miner undergoing a left pneumonectomy for carcinoma who developed a cardiac arrest on the operating table. The remainder of the complications were reversible and correctible. The incidence of postoperative bronchoscopies for retained secretions was 10 per cent, which is about the same as in our control series with conventional endotracheal anesthesia. No cerebral symptoms were detected that could be ascribed to prolonged hypoxia.

SUMMARY

A series of 200 consecutive patients undergoing intrapleural surgical procedures have been intubated and ventilated by means of a Carlens endobronchial catheter. All operations have been performed with the patients in the lateral position. The operative lung has been allowed to become acutely atelectatic during the hilar dissection and bronchial stump closure. The patient is maintained on the contralateral lung during the period of acute atelectasis.

Animal experiments on 6 mongrel dogs indicate a mean drop of 10 per cent in arterial oxygen saturation when the operated lung is atelectatic for 30 minutes. With additional occlusion of the pulmonary artery to the operated lung there is a mean rise of 6 per cent in arterial oxygen saturation. Arterial blood pH and carbon dioxide content remained fairly constant during this period of occlusion.

CONCLUSIONS

1. The Carlens double-lumen endobronchial catheter is a useful instrument in the surgical management of intrapleural conditions.

2. The advantages of better surgical exposure, an immobile operative field, easier hilar dissection, and better management of the bronchial stump outweigh any theoretical disadvantages.

3. The anesthesiologist has better control of the situation in the event of sudden hemorrhage or copious bronchial secretions.

4. The venoarterial shunt created by controlled atelectasis of the operative lung is not physiologically significant if the patient is in the lateral position and the down lung is used to the maximum with controlled respiration and ventilated mechanically.

5. The routine use of the Carlens endobronchial catheter for adult pulmonary surgery is recommended.

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DISCUSSION

DR. CLARENCE CRAFOORD, Stockholm, Sweden.—I can only underline that we have had experience in using the Carlens catheter in about 2,000 cases up to this time and we have met in a few instances these same difficulties that the previous speaker has described. But, on the whole, we think it is a very useful tool, especially in cases where you can expect to have bleeding or a lot of secretion from the lung under operation.

DR. WARREN J. TAYLOR, Boston, Mass.—For years our service at the Peter Bent Brigham Hospital has enjoyed a close relationship with the Veterans Hospital at Rutland Heights, Massachusetts, where there has been extensive experience with the Carlens catheter. This started in 1950 when Dr. Crafoord visited that hospital and suggested to Dr. Harken and Dr. John Cincotti, then the Chief of Surgery, that the Carlens catheter be used.

The Carlens catheter has now been used in approximately 1,100 cases. We agree entirely with Dr. Newman that complications and difficulty of intubation are not unduly troublesome. We have achieved good oxygenation.

Our chief difficulty has been with the anesthesiologists who, for various reasons, didn't care to use the catheter. The complaint of difficulty in intubation was corrected by demonstrating the technique of insertion.

[Slide] There was genuine concern about increased airway resistance. The rotameter studies shown on this slide demonstrate that there is not a great deal of airway resistance with the Carlens catheter. At a flow rate of 40 L. per minute, through either the right or left side, there was less than 10 cm. of water pressure differential. Through the combined orifices, the resistance is about 3 cm. These results compare very favorably with a No. 7 McGill single lumen tube and a 32 Fr. tube. Thus, resistance is no problem.

A second objection has been that badly distorted tracheas will not accept the Carlens catheter. This can very well be the case, as Dr. Newman has pointed out, but it is not always so. Dr. Thomas Enright, currently Chief of Surgery at Rutland Heights, made these slides available. This slide shows a badly distorted trachea which deviates markedly to the left of the midline in an individual with a totally destroyed left lung. A Carlens catheter was readily introduced into this trachea. The next slide shows the catheter in place and the trachea straightened out very nicely. This isn't always possible, but very often it is. We do not think that distortion of the trachea per se should dissuade one from using the catheter in view of the many other advantages.

DR. JOHN J. CINCOTTI, Albany, N. Y.—I do not believe that routine use of this catheter will become very popular because of the difficulties in obtaining the eager cooperation of the anesthesiologist and, second, the rare difficulties with intubation, especially crossed bronchial intubation.

Some 11 years ago, following Dr. Crafoord's visit with us at the VA Hospital, Rutland, Massachusetts, we began to use the Carlens catheter routinely as an anesthesia tube during the gamut of thoracic surgical procedures. Our main interests were to observe the extent of pulmonary function loss during the operative procedure and to determine the end-operative distribution of function of the lungs (Watson et al., Am. Rev. Tuberc. 75: 730, 1957); what factors tended quickly to return the remaining parenchyma to full function, and what factors (the tight dressing) worsened function, and exactly what the proper management of the pleural space might be.

[Slide] This slide shows what happens during the simple operation of phrenic nerve crush. Recall that in that era some advocated post-resection crush or blocking of the phrenic nerve in order to reduce the extent of the pleural cavity so as to approach the volume of remaining lung. In a group of 11 patients, 9 exhibited the ordinary unilateral function loss after phrenic crushing. However, in 2 of the 11, there was a dramatic bilateral ventilatory loss. It has long been known that there exists terminal branch crossings of the phrenic nerves from one side to the other, in man. Prior to our observations, a single instance of functional crossed *peripheral* innervation was reported, and this in the cat (Rosenbluth). We think it is worth knowing that ipsilateral function loss follows phrenic nerve crush and, on occasion, the defect may be bilateral.

[Slide] This shows the end-operative status of the patient following unilateral thoracotomy. The first three strips show what usually happens and, as you see, the first and third strips are quite comparable, showing that after the removal of nonfunctioning, diseased tissues, there is virtually no measurable function loss. However, the next three strips show that there may be unexpected and significant changes; that on occasion the lung will act as though a unilateral apneic oxygenation is occurring, or again, as in the next to the last strip, that a tracing, which in all respects resembles that found in agenesis of a pulmonary artery, is observed. The last strip depicts a "functional pneumonectomy" tracing following a subtotal pulmonary resection, end operatively. The losses are temporary but unpredictable; full return of these measures of function may require weeks, even in the absence of clinical complications.

[Slide] One observation that grew out of this study is the importance of the degree of negative breaking suction used in pleural space drainage systems. The literature that refers to it uniformly suggests – 8 or – 10 cm. of water negativity. This slide documents the fact that, in the end-operative period and probably in the postoperative period, the patient has a much wider excursion than this and if you regulate the negative suction system so that only 10 cm. is obtainable, you actually induce hypoventilation of that lung. This defect is eliminated by increasing the negativity to 18 to 20 cm. H_2O.

[Slide] Here is shown the type of valve devised for the Carlens catheter for this study (Anderson and Cincotti, J. Lab. & Clin. Med. 45: 493, 1955). This is a very lightweight valve that allows instantaneous changing from either orifice of the double-lumened catheter permitting unilateral toilet, administration of anesthetic mixtures, deflation, inflation, and discontinuous bronchospirometry.

DR. JOFFRE-ANDRE GRAVEL, Quebec, Quebec.—Having trained with Dr. Crafoord, it came about quite naturally that we should use the Carlens tube, especially after having encountered Dr. Carlens, and this was a charming encounter, I assure you. I would like to show you quickly three slides of our experience with the use of this tube over the past 2 years. [Slide] The tube has been used in about 1,200 resections, mostly for tuberculosis. From 1951 to 1956 we found the use of the tube necessary in about 42 per cent of these operations, but as we got wiser surgically, I suppose, the use of the tube became not quite so necessary and we were able to bring the percentage down to 27.

[Slide] The advantages we found are classified here: ventilation and aspiration can be easily done in wet cases, deflation and inflation of the lung is better facilitated; and there is better maintenance of anesthesia.

[Slide] One little drawback that we have found has been postoperative edema of the bronchus when possibly the balloon has been overinflated. This, however, has not been too severe and has responded very well to the use of steroids.

Occasionally, it has not been easy to put in the tube when the trachea has been modified, but, all in all, we have been pleased with the tube and think it has, at least in our early days, helped us tremendously in our surgery.

DR. EDWARD A. GAENSLER, Boston, Mass.—The first, and perhaps the most important, is to emphasize two words in the title: "routine use." You have heard today a number of series reported in which this catheter was used more or less routinely and results were excellent. I should like to warn against limiting the use of this catheter only to cases where it is essential and absolutely indicated. Such cases may not occur more than once or twice a year—multiple lung abscesses, massive bleeding at the time of operation. If a bronchospirometric catheter is used only on such occasions (and several such instances have come to my attention), then often neither the anesthetist nor the surgeon is familiar with the technique and additional difficulties are added in an already difficult situation, and the results may be disastrous.

Therefore, some sort of routine use in patients in whom the catheter is not essential is important so that the entire team will become familiar with it. Whether this routine use be for all lung cases, or all patients with lung abscess, or all patients over 65 makes little difference.

The second point I would like to make is that an excellent way of introducing this catheter is by topical anesthesia with the patient in the sitting position. We have intubated in this way in about 2,400 cases and have failed on only three occasions.

The third point I would make is that the significance and importance of arteriovenous shunt during atelectasis with open pneumothorax is quite controversial. As soon as the lung collapses, airway resistance increases and the lung becomes atelectatic; but, also, the resistance to blood flow increases greatly and very quickly nearly all the cardiac output is shifted to the opposite lung. Those of you who may have had some contact with pneumothorax therapy will recall that, on occasion, inadvertently a total pneumothorax

was produced during a "refill." Usually, neither the patient nor the physician was aware of this accident at the time, cyanosis was not visible, and oxygen saturation often was normal.

DR. DOWNS (Closing).—I wish to take this opportunity to thank the Association for the privilege of the floor and I would also like to thank those individuals who were kind enough to comment upon this paper. In fact, I am a little surprised that the comment was so favorable. I rather expected to be taken heartily to task with regard to the venoarterial shunt problem.

I think our argument revolves around one very important point, and that is the question of over-all safety of the patient during thoracic surgery. We believe that the routine use of the Carlens catheter provides the surgeon with the most ideal operating field. We thereby materially reduce the opportunity for surgical accident, and, therefore, give the patient a better chance for an over-all successful result.

With regard to Dr. Gaensler's remarks about the significance of blood shunting, I believe that currently there is some argument as to whether blood is shunted away from the atelectatic lung or not. It would be our opinion that, initially during atelectasis, the blood flow through the operative lung is probably pretty close to normal.

If we contemplate the oxygen uptake in the ventilated lung, we can easily see how we can put in, without fear of deficit, the amount of oxygen which the patient is utilizing under anesthesia. Furthermore, on the basis of our experience, altitude anoxemia studies, and the well-known oxygen dissociation curve, it is my opinion that safe tissue oxygenation can be maintained in the presence of arterial oxygen saturation levels of 80 per cent.

Anemia and hypovolemia preclude complete unilateral induced atelectasis.

I think evolving to the point where one advocates routine use of the tube is a long-range process, but that it is well worth the effort in terms of the final outcome in the patient.

COMPLETE FUNCTIONAL RESTITUTION OF THE FOOD PASSAGES IN EXTENSIVE STENOSING CAUSTIC BURNS

Joseph H. Ogura, M.D. (by invitation),*
*Charles L. Roper, M.D.** (by invitation), and*
*Thomas H. Burford, M.D.,** St. Louis, Mo.*

In 1951, Bosher and Ackerman and one of us (T. H. B.)[1] reported on experimentally produced caustic burns of the dog's esophagus and stressed the irreversibility of these lesions. In 1953, Webb and Ackerman and one of us (T. H. B.)[2] presented a series of patients treated by excision of the stenosed esophagus, elevation of the stomach into the thorax or neck, and esophagogastric anastomosis. The uniformly excellent results, when contrasted with the ordeal of repeated dilatations, led us to recommend early excision and surgical restoration of deglutitory adequacy and continuity in all established strictures.

Despite assent from some, increased experience by ourselves and others has strengthened the validity of our recommendations. While stomach substitution served admirably, the possible advantages of jejunum or colon are conceded. Experience has equilibrated the choice, in our opinion, in favor of the right colon in benign obstruction, except when stenosis is limited to the lower third of the esophagus. In this group, we prefer jejunum.

The caustic stricture, which spares the pharynx and cricopharyngeal pinchcock, is now said to be a safely correctable lesion. There are, however, a number of cases in which the burn and subsequent stricture involves the pharynx, epiglottis, and cricopharyngeal pinchcock, as well as the esophagus. Heretofore, these individuals have of necessity been committed to a lifetime of gastrostomy or jejunostomy feedings.

Observations made on 65 cancer patients in whom larynx-sparing operations were performed have revealed that certain structures may be sacrificed without compromising function. These structures are the epiglottis, aryteno-epiglottic folds, false cords, the greater part of the hypopharynx, and one arytenoid cartilage. The superior laryngeal nerves, part of the cricopharyngeal pinchcock, and the base of tongue may also be sacrificed. These facts established by one of us

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(J. H. O.)[3-6] suggested that the principles of this operation, combined with a modified right colon transplant, would re-establish normal deglutition in a patient with extensive stenosis of the pharynx and esophagus.

HISTORICAL REVIEW OF ESOPHAGEAL SUBSTITUTION

Kelling[7] and von Hacker[8] were the first to describe the use of the colon for esophageal replacement. Since 1911 there have been numerous publications[9-14] suggesting colon replacement for a variety of esophageal lesions. There have been no reported series of cases in the literature in which colon substitution combined with reconstruction of the pharynx has been successfully accomplished for extensive caustic burns.

TECHNIQUE

At the time of anesthesia induction the presence of marked pharyngeal scarring has presented difficult technical problems in endotracheal intubation. We believe that tracheostomy should be avoided during the first-stage procedure.

In this series of patients, the operation has been staged to minimize possible contamination of the anterior mediastinum. Infection may occur as a complication of tracheostomy—a necessary technical step for laryngeal and pharyngeal reconstruction. As our experience increases, we feel that a one-stage procedure may be readily performed.

First Stage.—

Preparation of the colon segment: Simultaneously, while the anastomotic site of the hypopharynx is exposed by a second operating team, the abdomen is opened through a midline incision extending from the xiphoid process to below the umbilicus (Fig. 1, *A*). The usual technique of right and transverse mobilization is executed. Inspection of the mesocolon is necessary to determine the presence of an adequate marginal artery and the sites of origin of the middle, right, and ileocolic arteries. For the usual colon bypass to the cervical esophagus, we have been able to include in the vascular pedicle both the middle and right colic arteries when the latter arose as a separate branch from the superior mesenteric artery (Fig. 1, *B*).

In this series of patients, however, it has been necessary to ligate both the ileocolic and right colic branches at their origins so that a greater length of colon could be obtained. Prior to ligation of these vessels, they are temporarily occluded by means of arterial clamps for a period of at least 10 minutes. During this interval, appendectomy is performed and the ileum is transected 1 cm. proximal to the ileocecal valve (Fig. 1, *C*).

The stump of the ileum is closed by one layer of continuous inverting 3-0 chromic catgut and by one layer of interrupted 4-0 black silk sutures. Inclusion of any portion of the cecal wall is avoided so that there will be no disruption of the intramural blood supply in this critical area.

Following ligation and division of the arterial branches, the colon segment is handled cautiously, since the avoidance of small subserosal and submucosal hematomas is mandatory. The colon segment is then passed posteriorly to the stomach and through an incision in the gastrohepatic omentum (Fig. 1, *D*).

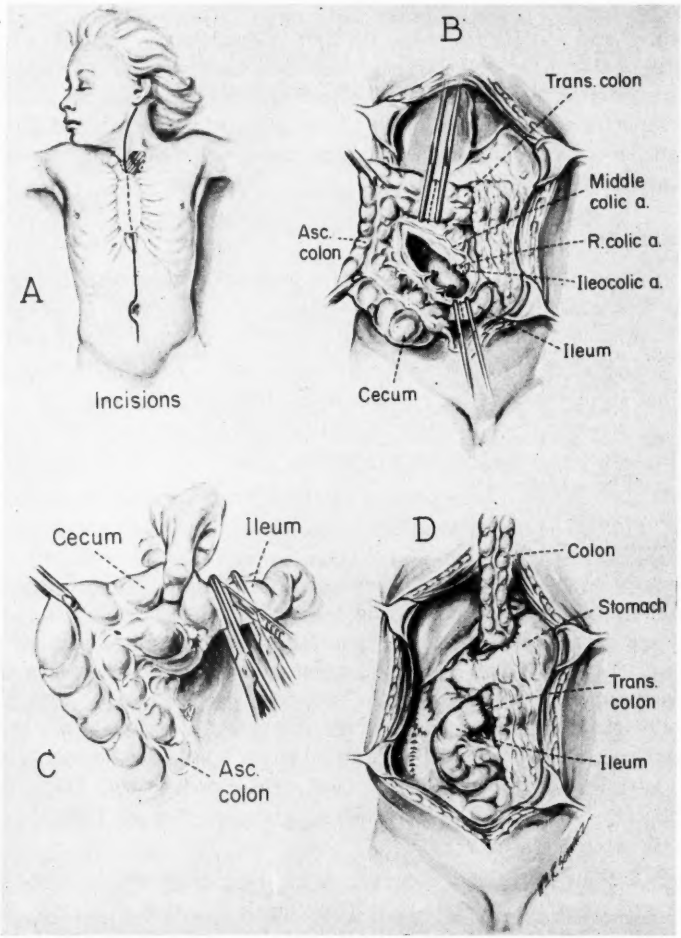

Fig. 1.—Preparation of the colon segment. *A*, Usual incision for colon substitution. (*Dotted line* indicates median sternotomy.) *B*, Right colon mobilized and the right colic and ileocolic arteries have been ligated and divided. *C*, Ileum divided 1 cm. proximal to ileocecal valve. *D*, End-to-end ileotransverse colostomy and cologastrostomy completed with vascular pedicle lying posterior to the stomach.

A median sternotomy is performed to allow precise placement of the colon segment within the confines of the anterior mediastinum. Twisting and undue tension of the vascular pedicle are to be avoided. A segment of clavicle and sternum on the side of the cervical anastomosis is resected to prevent significant compression of the colon in the region of the thoracic inlet.

The transverse colon is divided at its proper site and, subsequently, an end-to-end ileotransverse colostomy is performed while the cecum is being anastomosed to the hypopharynx by the other operative team.

A cologastrostomy is then performed on the anterior surface of the stomach. Although one strives to place the anastomosis high on the fundus of the stomach,

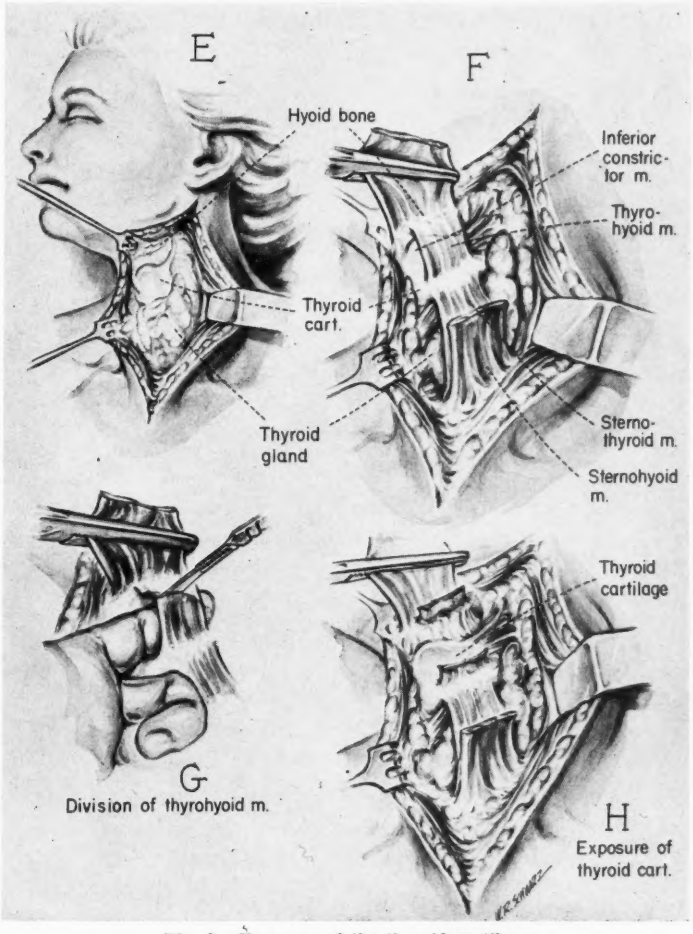

Fig. 2.—Exposure of the thyroid cartilage.

extreme angulation of the distal colon segment must be avoided. This may be partially overcome by excision of the xiphoid process and by a 2 cm. incision in the diaphragm. To obtain a satisfactory anastomotic site, it has never been necessary to take down the gastrostomy which may have been present for several years.

Preparation of the anastomotic site of the hypopharynx: Selection of the side of the pharynx for anastomosis is determined by direct laryngoscopy, and

the pyriform sinus with the least scarring is used. The skin incision is made along the anterior border of the sternocleidomastoid muscle from the angle of the jaw to the suprasternal notch (Fig. 2, *E*). The skin flaps and subcutaneous tissues are elevated over the larynx and dissection is carried toward the posterolateral border of the thyroid cartilage. The sternohyoid and thyrohyoid muscles

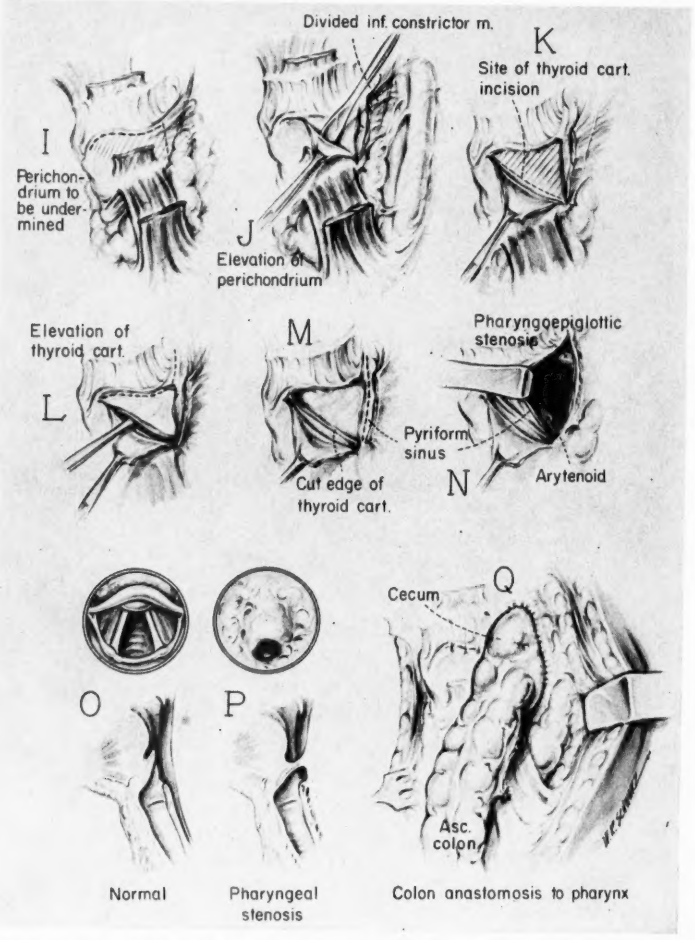

Fig. 3.—Exposure of the anastomotic site of the pyriform sinus. *O*, Normal view. *P*, Note the diaphragm-like pharyngeal stenosis. The arytenoid cartilages are visualized distal to this small opening.

are divided transversely at the superior border of the thyroid cartilage (Fig. 2, *F, G, H*) and the medial constrictor muscles are divided along the posterolateral border of the thyroid cartilage. From the thyroid notch to the superior cornu, the perichondrium is incised, and the external perichondrium is elevated inferiorly (Fig. 3, *I, J*). The cartilage is incised obliquely from the thyroid notch

to a point just superior to the inferior cornu (Fig. 3, *K, L*). Resection of this cartilage will expose the internal perichondrium and the hypopharyngeal area (Fig. 3, *M*).

The pyriform sinus will be seen bulging at the posterolateral border of the thyroid cartilage. When the cecum is presented for anastomosis, the hypopharynx is opened vertically, and one arytenoid eminence is identified (Fig. 3, *N*). The incision is then extended superiorly to the point where the epiglottis and pharynx are bound laterally and posteriorly; this will create a substantial

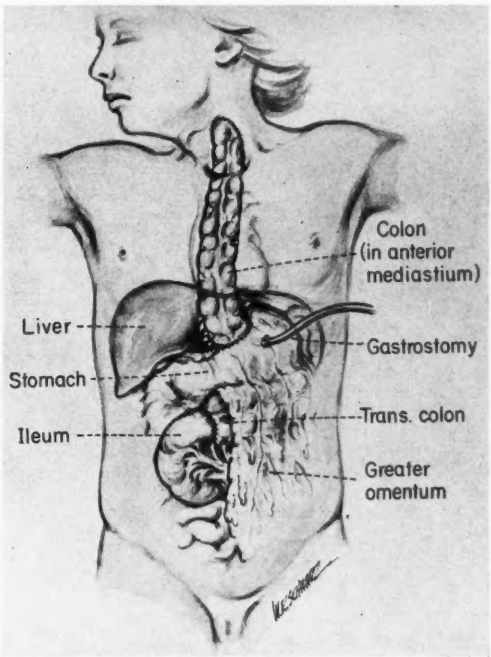

Fig. 4.—Schematic view on completion of the first stage of colon substitution to the pharynx.

pharyngostoma (Fig. 3, *N, O, P*). The cecum is now opened along a taenia for a distance of 3 cm. and an end-to-side anastomosis is made with the hypopharynx, using a single layer of interrupted 4-0 arterial silk (Fig. 3, *Q*; Fig. 4).

Drains are placed into the neck and upper mediastinum and the subcutaneous tissues and skin are then closed with interrupted silk.

Second Stage.—

Two weeks after the colon substitution has been performed, the patient is prepared for the second phase. A preliminary paramedian tracheostomy is performed through the second and third tracheal rings under local anesthesia and endotracheal anesthesia is then administered. Such a paramedian tracheostomy will lessen accidental exposure of the colon segment in the opposite side of the neck.

A wide transverse incision is carried across the hyoid bone and dissection of the skin flaps is carried for a short distance inferiorly to the region of the superior border of the thyroid cartilage. The hyoid bone is split in the midline with partial separation of the suprahyoid muscles (Fig. 5, *A*, *B*). As dissection is carried into the pharynx, the mucous membrane of the vallecula will be identified.

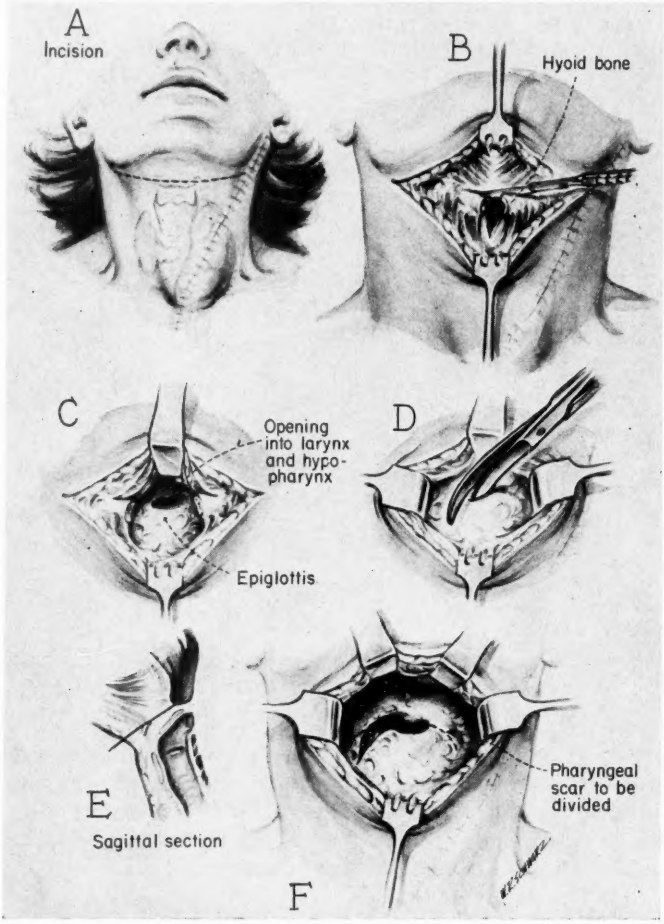

Fig. 5.—Reconstruction of pharynx. *A* and *B*, Exposure of the hypopharynx by the transhyoid approach. *C*, Pharyngo-epiglottic stricture. *D*, *E*, and *F*, Division of epiglottis from lateral pharyngeal wall.

Immediately upon entering the pharynx, the diaphragm-like epiglottic-pharyngeal scar will be identified (Fig. 5, *C*). For additional exposure a midline extension is made inferiorly down to the thyroid notch.

The scarred supralaryngeal structures are excised. The epiglottis will be attached posteriorly by a dense broad band of scar tissue. The margins of the epiglottis are cut on each side but great care is exercised since normal landmarks are absent (Fig. 5, *D*). The region of both arytenoids is next visualized

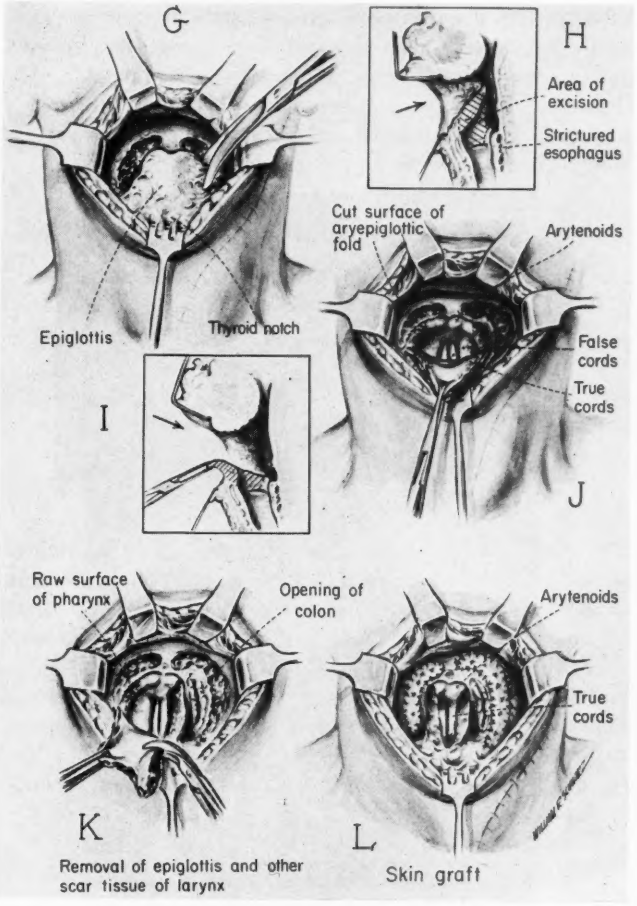

Fig. 6.—Pharyngeal reconstruction by resection of supraglottic structures and skin graft. *H* and *I*, The *dotted lines* indicate the area of excision. *K* and *L*, Note that the arytenoid cartilage must not be exposed and that a cuff of mucosa is preserved about the colon stoma.

and one must avoid accidental incision into the colon as the lateral glossoepiglottic structure is released from the pharyngeal wall (Fig. 5, *E*, *F*; Fig. 6, *G*, *H*, *I*). The arytenoid cartilage should not be exposed, otherwise fixation of the cord will result. Impairment of one arytenoid will result in a hoarse voice, but bilateral fixation will present a serious problem with tracheostomy decannulation.

The scarred aryepiglottic fold is cut above the arytenoid cartilage and the dissection is carried into the endolarynx to below the level of the false cords (Fig. 6, *J, K*). As one dissects with a curved scissors into the anterior pyriform sinus, the close proximity of the cut margin of the hypopharyngeal mucosa to the colon lumen will be evident. Great care is exercised to preserve a cuff of mucosa at the previous pharyngocecal anastomosis.

The entire epiglottis is then removed from beneath the thyroid notch together with the opposite false cord and aryteno-epiglottic fold. At this point, one should lighten the depth of anesthesia to determine the function of both true cords. It will now be observed that both pyriform sinuses and the greater part of the hypopharynx will be devoid of mucosa.

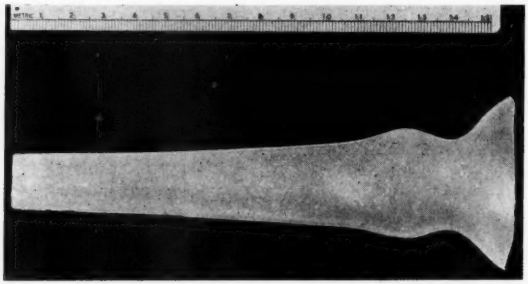

Fig. 7.—Negus stent.

A split thickness skin graft from the inner aspect of the thigh is taken and placed into the pharynx where it is held with catgut mattress sutures (Fig. 6, *L*). The skin graft is approximated closely to the small strip of mucosa along its posterior pharyngeal wall, and to the mucosal strip about the colon stoma.

If webs of the colon stoma are noted they are divided and an appropriate-sized Negus stent is placed into the colon (Fig. 7). This stent which assists in securing the skin graft to the pharynx should be anchored by a silk suture to the nose to prevent its accidental descent into the colon. The hyoid bone is sutured together, and subcutaneous tissues and skin are approximated with interrupted silk sutures.

RESTITUTION OF DEGLUTITION

Besides the routine postoperative care and feeding per gastrostomy, considerable attention must be given to tracheostomy care. The indwelling Negus stent is maintained for 2 or 3 weeks. Upon removing the stent, indirect laryngoscopy is performed. If the edema has sufficiently subsided, the patient may attempt deglutition.

The pharynx must be healed and the airway re-established through the larynx before deglutition is attempted. Difficulty with deglutition may last from several days to 3 weeks. Aspiration of liquids is the general rule with the first attempts at ingestion. The patient must make a conscious effort to clear the airway simultaneously with the effort to move food into the colon. Semisolid

foods are the first to be tolerated. The tracheostomy tube must be corked at this point to use the expiratory air column to assist in clearing the larynx. During the rehabilitation period when liquids appear to be swallowed with some difficulty but solids reasonably well, the tracheostomy tube is removed. Rapid restitution to normal unconscious deglutition will be dramatic.

PRESENTATION OF CASES

Six patients with extensive stenosis of the pharynx and esophagus were operated upon. Of the 6 operations, 5 were completely successful. The only postoperative death occurred in a 2-year-old boy. Five patients had ingested

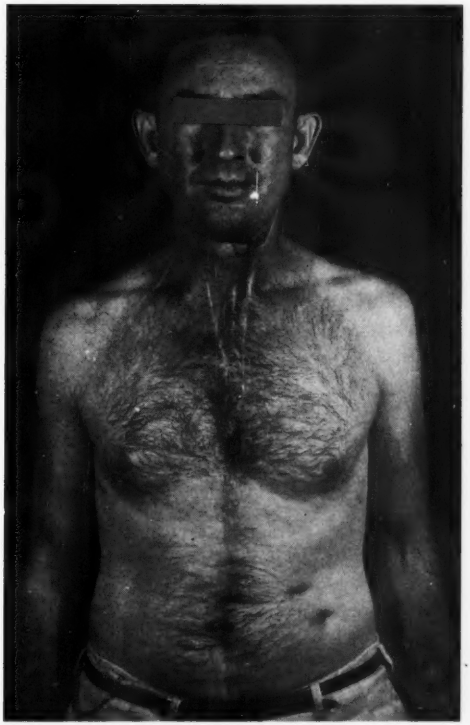

Fig. 8.—Photograph made 19 months postoperatively of patient who had the 2-stage operation.

lye and one sulfuric acid. All showed extensive cicatricial stenosis of the pharynx with almost complete closure of the esophagus necessitating gastrostomy for feeding purposes. None could swallow liquids or saliva and aspiration of pharyngeal secretions was common.

In these instances the use of steroids and antibiotics had failed to halt stricture formation. The time interval between ingestion of the caustic substance and the first stage of the operation varied from 9 months to 6 years.

All 5 patients, who were followed for a period as long as 1½ years, manifested excellent deglutition and voice function. No postoperative dilatations have been necessary. It is significant that these patients ingest all types of foods and have gained weight (Fig. 8). Accidental aspiration has not occurred.

DISCUSSION

Although this reported series of patients represents the most advanced stage of pharyngeal and esophageal stenosis, it is well to point out that lesser degrees of pharyngeal involvement may be easily overlooked at the time of esophagoscopy. In these instances, when the usual substitution procedures are performed for an obvious lesion of the esophagus itself, normal deglutition will not be established. Thus accurate preoperative evaluation is of paramount importance.

We would also stress the importance of preoperative bowel preparation in all colon substitution procedures. The use of intestinal antibiotics (Sulfasuxidine together with neomycin) must be combined with thorough mechanical cleansing measures (castor oil and enemas).

Meticulous attention should be given to technical detail in all stages of colon substitution if fistulas and subsequent strictures are to be avoided.

This operation is applicable to situations other than strictures due to caustic substances. One 48-year-old woman with a 2 year history of symptoms, and not included in this series, presented with a long post-cricoid stricture of the esophagus which was complicated by a tracheoesophageal fistula and complete bilateral vocal cord paralysis.

A previous attempt at local repair was unsuccessful. In this patient, the cecum was anastomosed to the pharynx and a secondary plastic repair of the vocal cords was performed. While hoarseness of voice has persisted, deglutition has been excellent.

Extensive cinefluoroscopic studies* and observations of patients in whom the supraglottic structures and part of the cricopharyngeus have been excised demonstrate that their removal does not impair deglutition. The total absence of a pinchcock in this series of patients confirms our conclusion that this structure is not necessary for satisfactory swallowing.

From this reported series of patients with pathologic derangement previously considered hopeless, it is evident that such a situation may be overcome by combined efforts. The success of this operation should be measured in terms of complete restitution of function. A most gratifying dividend in these patients has been the return to a normal psychologic and sociologic state.

CONCLUSION

A series of patients with extensive stenosing caustic burns of the pharynx and esophagus have undergone a combined operative procedure consisting of esophageal substitution by the right colon together with plastic reconstruction of the pharynx. The results have been uniformly successful.

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(For Discussion, see page 359.)

INCOMPETENCY OF THE GASTRIC CARDIA WITHOUT RADIOLOGIC EVIDENCE OF HIATAL HERNIA

The Diagnosis and Management of 71 Cases

Clement A. Hiebert, M.D. (by invitation), and*
*Ronald Belsey, M.S., F.R.C.S.** (by invitation), Bristol, England*

IF one accepts the thesis, convincingly argued by Allison[1] 10 years ago, that the essential feature of a sliding hiatal hernia is a valve failure at the cardia and that its symptoms derive from gastric fluid escaping into a sensitive esophagus, then the term "hiatal hernia" is unfortunate. For the suggestion is implicit that the protruding stomach is what matters; particularly if it is small it is apt to be dismissed by the radiologist as "of no clinical significance." Yet in the course of 640 operations for hiatal hernia at Frenchay Hospital it has become increasingly evident that the size of the hernia has little correlation with the patient's complaints; often the smaller the hernia the more incompetent the cardia and the more distressed the patient.

Furthermore, it has emerged that severe symptomatic gastroesophageal reflux may exist in the complete absence of a hiatal hernia when the barium studies are "normal," despite the best efforts of the radiologist to coax stomach above the diaphragm. This isolated defect of the hiatal valve mechanism and its surgical treatment was first recorded by Belsey[2] in 1952. Two years later the same idea was treated philosophically by Barrett.[3] Stensrud,[4] in 1957, described successful surgical treatment of one patient. More recently, Cross, Smith, and Kay[5] have reported reconstruction of the cardia in 7 patients with esophagitis but no hernia.

The purpose of this paper is to discuss the diagnosis, treatment, and follow-up study of 71 patients treated between the years 1951 and 1959.

THE CLINICAL PICTURE

Diagnosis is established, first, by the symptoms, which are identical to those of a sliding hernia (Table I), and second, by the discovery of patulous cardia during esophagoscopy.

Read at the Forty-first Annual Meeting of The American Association for Thoracic Surgery at Philadelphia, Pa., April 24-26, 1961.

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TABLE I. INCIDENCE OF SYMPTOMS OF ADULT PATIENTS WITH PATULOUS CARDIA AND GASTROESOPHAGEAL REFLUX

Postural aggravation	70	(99%)
Epigastric pain	57	(80%)
Regurgitation	55	(77%)
Heartburn	51	(72%)
Back pain	38	(54%)
Chronic vomiting	36	(51%)
Sleepless nights	36	(51%)
Dysphagia	32	(45%)
Dry mouth	14	(20%)
Choking feeling or dyspnea on lying flat	9	(13%)
Flatulence	8	(11%)
Hematemesis	5	(7%)
Night cough relieved by operation	4	(6%)
Fatty food intolerance	3	(4%)
Neck pain	3	(4%)
Otalgia	2	(3%)
Pain in arm or wrist	2	(3%)
Burning nose and filthy tongue	1	(1%)

This report will tend to emphasize the more severe features, for all 71 patients reached this thoracic service by referral from other doctors, frequently after an unsuccessful trial of therapy ranging from abdominal laparotomy to psychotherapy. The mean duration of symptoms for the adult group was 5 years. Fifty-six patients had been on a medical program, usually based on a bland diet plus antacids. Ten unsuccessful operations had previously been performed elsewhere for the same complaints. These are enumerated in Table II.

TABLE II. OPERATIONS DONE PREVIOUSLY FOR THE SAME COMPLAINTS

Subtotal gastrectomy	1
Ramstedt operation	1
Exploratory laparotomy and appendectomy	3
Cholecystectomy	3
Right upper quadrant laparotomy only	2
Total	10

A history of *dysphagia* was present in half of the group. Although this is the expected symptom with the complication of benign stricture, the chronic flooding of the lower esophagus with gastric enzymes may wreak havoc with swallowing well before stenosis is apparent on the x-ray film or esophagitis is visible by esophagoscopy.

Half of the total number of patients complained of *sleepless nights* because of regurgitation, heartburn, or chronic night cough due to unconscious aspiration of stomach contents flooding the pharynx. Confirmation of this explanation was suggested by the immediate disappearance of this cough after operation. Altogether 15 patients showed clinical, radiologic, or operative evidence of aspiration pneumonitis.

Chronic vomiting was a complaint of 51 per cent of the patients and probably reflects the inability of the valveless stomach to retain food or fluid under pressure. Usually there is no true nausea prior to the act of vomiting.

One patient (C. G., No. 49612) was a 38-year-old man who had regurgitated after every meal for as long as he could remember. In order to remain alive, let alone socially acceptable, he had to hold the vomitus in his mouth until he could reswallow it. This ability to ruminate earned him understandable notoriety as a child when, to the amusement of playmates on a bridge above the train tracks, he would vomit at request upon the engines passing beneath. There was no hernia by x-ray examination, but esophagoscopy revealed a typical patulous cardia. Restoration of a normal valvular mechanism promptly ended his regurgitation.

Whereas *epigastric pain, flatulence, heartburn, vomiting,* and *back pain* are symptoms common to a number of upper abdominal conditions, the pathognomonic hallmark of an incompetent cardia is the history of *postural aggravation* of symptoms, usually an increase of epigastric discomfort or substernal burning when bending or stooping. One patient recalled severe burning in her pharynx and nose when leaning over the wash bowl to brush her teeth. Others complained of precordial suffocation when lying flat. (Fabricius[6] cites Galen as naming the uppermost valve of the stomach, "the heart" or "cardia" because of the similarity of symptoms originating here to those arising from the heart!) In the differential diagnosis the feature of postural aggravation cannot be overstressed. It must be sought for with the same directed interest with which one pursues the clue of epigastric pain relieved by alkali or food in a patient suspected of peptic ulcer disease.

ESOPHAGOSCOPY

Esophagoscopy is as indispensable in the evaluation of any patient suspected of a patulous cardia as cystoscopy is in the study of a patient with disease of the bladder. Not only is esophagoscopy the confirmatory test for the entity of gastroesophageal reflux, but it is the only certain way to assess the presence of esophagitis in its earlier stages.

In adults, the examination is preferably done under local anesthesia in order to gain the maximum information regarding the status of the diaphragmatic part of the valve-closing mechanism. Normally with the patient relaxed in the sitting position, the cardia presents a rosette of converging esophageal mucosa, ascending and descending with movements of the diaphragm but remaining closed throughout the respiratory cycle. Further advance of the esophagoscope is limited.

In the patient with an incompetent valve, on the other hand, vertical movements of the lower esophagus with respiration are diminished or absent. There is no descent of the cardia with inspiration. Instead of the usual tight constriction it gapes widely when the patient takes in a deep breath and indeed may remain open during expiration as well. Frequently one may look directly into the stomach. Alternation of pressure differences between the stomach and esophagus are manifest by an obscuring tide of gastric fluid rising and falling with each breath. The deleterious action of this enzymatic ebb and flow was evident in a third of the patients who showed various stages of reflux esophagitis.

X-RAY FINDINGS

None of the 71 patients had a demonstrable hernia by x-ray studies, but, in 26, barium was seen to reflux into the esophagus. Usually this finding was associated with widening of the cardia, a point illustrated by Figs. 1 and 3. Perhaps the most impressive feature of the x-ray studies is the thickness of the envelope containing "negative" barium films.

Sweet[7] has underlined the importance of informing the radiologist, when a hiatal hernia is suspected, as his zeal is an important factor in persuading barium to outline a small hernia. The method of examining patients at Frenchay Hospital has been described[8] and includes maneuvers in the erect, supine, oblique, head-down, and stooping positions. Using these techniques it has been possible to increase by 12 per cent the incidence of hiatal hernia in a group of 333 patients who had been first examined elsewhere.

TREATMENT

When a patient presents with a history suggestive of gastroesophageal reflux and esophagoscopy confirms the presence of a patulous cardia, surgery is advised only if the symptoms are intolerable or have persisted under medical treatment. On the other hand if the finding is associated with esophagitis, chronic blood loss, anemia, or aspiration pneumonitis, operation is recommended even when subjective complaints are minimal. In the experience of this service, esophagitis usually is a progressive disease which is unlikely to heal permanently on medical management; while waiting for the outcome of conservative therapy irreversible damage leading to stricture may occur.

Except in the presence of periesophagitis or stricture, the appearance of the esophagus and cardia at thoracotomy is disappointingly normal. The essential pathologic change is not to be seen from the exterior but will have been observed beforehand through the esophagoscope. A reasonably consistent finding at operation is laxity of the hiatal muscular sling. After the attachments of the lower esophagus had been freed up, traction on the esophagus was seen to produce a small hernia in a quarter of the cases.

The operation is designed to eliminate gastroesophageal reflux by restoring a competent valve to the cardia. Details of the operative maneuvers have been reviewed elsewhere,[2] but the salient features of operation are depicted in Fig. 4 and are accomplished as follows: First, the lower attachments of the esophagus and cardia to the diaphragm are divided as if repairing a frank hernia. An acute angle of entry at the cardia is next created by placing mattress sutures between the gastric fundus and the anterior two thirds of the circumference of the lower 2 to 4 cm. of esophagus. As these are tied, the esophagus is partly intussuscepted into the stomach. This newly created angle is further reinforced and anchored within the abdomen by a second row of sutures, placed as before but also passing through the diaphragm, thus restoring an intra-abdominal segment of esophagus. Last, the effectiveness of the diaphragmatic pinchcock is provided by approximating the two halves of the right crus behind the

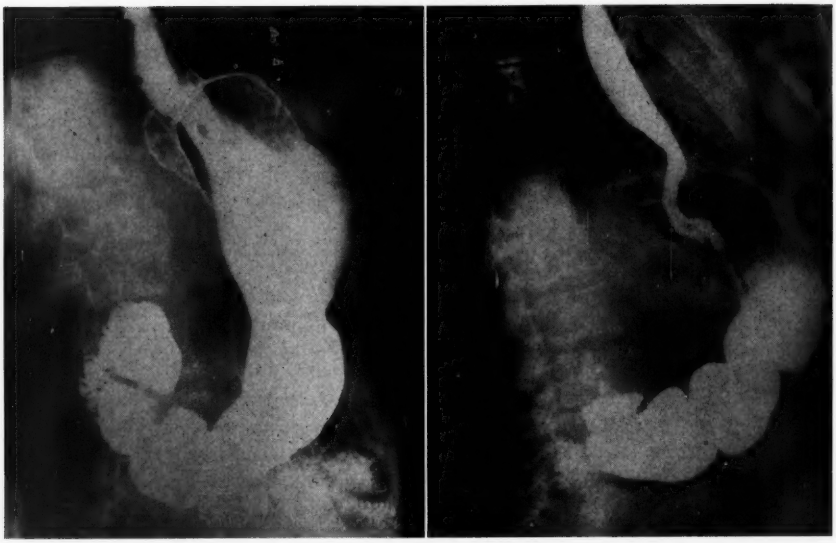

Fig. 1.—Preoperative barium study of a patulous cardia with marked reflux into the esophagus. A correct x-ray diagnosis was possible in slightly more than a third of the patients.

Fig. 2.—A postoperative barium meal which outlines the features schematically recorded in Fig. 4.

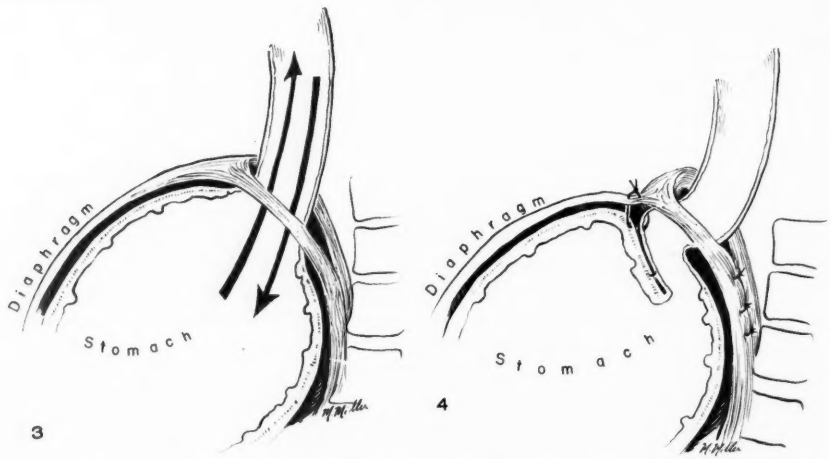

Fig. 3.—Schematic sagittal representation of a patulous cardia. The hiatus, formed by the two halves of the right crus, is lax and the esophagus joins the stomach like the neck of a bottle.

Fig. 4.—Diagram to show the reconstructed hiatus. There is now an intra-abdominal segment of esophagus, which will be collapsed against the crural buttress when the intraluminal pressure of the stomach exceeds that of the esophagus.

esophagus with deeply placed sturdy linen sutures. Tying these narrows the hiatus and provides a firm buttress against which the intra-abdominal segment of the esophagus will be compressed when the intragastric pressure rises (Figs. 2 and 4).

FOLLOW-UP STUDIES AND RESULTS

It is the practice here to follow every patient operated on for the remainder of his or her life. Patients with incompetent cardia repaired 8 years ago are still being seen at yearly intervals even though up to the present time they have been symptom free.

The follow-up period in the present group has ranged from 2 months to 8 years. Sixty-four patients (90 per cent) were personally seen in the clinic during the past year. Four more (5 per cent) were checked only by letter, questionnaire, or phone conversation. Three patients (4 per cent) are unaccounted for.

Of the patients treated surgically, good to excellent results were present in 54 (87 per cent). One of the interesting things about a successful operation is that the patient knows it from the first day he begins to swallow.

Four patients (6 per cent), while acknowledging improvement by surgery, did not warrant classification as good results. Another 4 were less than satisfactory.

By far the most frequent postoperative complaint is continued flatulence. Patients with gastroesophageal reflux are chronic air swallowers and this habit has to be unlearned. Before operation frequent swallowing is prompted by gastric fluid appearing in the pharynx. Following a successful repair of the patulous cardia, continued aerophagia is unfortunate, for the valve usually is competent to air as well as fluid.

Complications also included 4 adults who required postoperative stretching for transient dysphagia. Such cases usually represent too much enthusiasm on the part of the operator in lacing up the crural buttress posterior to the esophagus. It is very important to leave a little room for food to pass.

Four adults complained of persistent wound pain. There was one esophageal perforation during esophagoscopy; it was recognized and healed on nonoperative management. One patient developed a cerebral thrombosis and hemiplegia postoperatively, with survival. There were no wound infections of note. The only death in the series was a woman with biliary tract cancer. In retrospect she probably suffered from the effects of this malignancy at the time of her original operation.

DISCUSSION

Papers dealing with esophagitis and benign stricture usually include an assortment of cases in which no hernia could be demonstrated. To explain the pathologic changes in the esophagus, "chronic vomiting," or "trauma secondary to an inlying nasal-gastric tube" are invoked. What seems much more likely

is that an unsuspected patulous cardia was present. Such patients vomit chronically because food is ill-retained in a valveless stomach. Lying in bed simply makes matters worse and, if the patient is in hospital, a nasal-gastric tube is certain to be inserted by the first house officer who comes along. In retrospect it is easy to blame the tube and the vomiting.

It seems highly probable that the condtion of patulous cardia is a precursor of hiatal hernia. Notwithstanding occasional congenital defects seen in infants, one would postulate, on theoretical grounds alone, the existence of such a precursor. It is pertinent to note that the average age of the patients in this series is 45, approximately 15 years younger than the average seen with hiatal hernia. The sex ratio is the same; namely, 2 females for every male. Actually 2 patients followed in the clinic with known patulous cardias were found many months later to have developed hiatus hernias. Still another man (J. D., No. 52158), who had persistent symptoms associated with a chronic duodenal ulcer, had no less than six barium examinations in an attempt to elucidate the cause. After a number of years, he was referred for surgery and was found to have a small hernia.

Probably all 3 of these are examples of patulous cardias progressing to hernia en glissade. Skeptics will argue that in each instance the hernia had been present all along and that it was merely bad luck on the part of the radiologist in failing to pin down his mercurial quarry. Ideas become fossilized in names. The idea that the symptoms of hiatus hernia derive from the hernia, rather than incompetency of the associated patulous cardia, will probably persist until a more physiologically accurate term for this syndrome finds common acceptance.

SUMMARY

A clinical and pathologic entity is presented which consists of gastroesophageal reflux in the absence of a radiologically demonstrable hiatus hernia.

In reviewing 640 hiatal herniorrhaphy operations, 71 instances of this syndrome have been discovered; since 1951, 62 patients have been operated upon, with amelioration of symptoms in 58 (93 per cent).

The diagnosis is established by the clinical history, of which the most pathognomonic feature is postural aggravation of symptoms on bending or lying, plus the finding of a characteristically patulous cardia by esophagoscopy under local anesthesia.

Complications of this condition are identical to those observed with gastroesophageal reflux deriving from an established sliding hiatal hernia. These include inflammation, ulceration, scarring, and stricture of the lower esophagus with secondary blood loss anemia.

One fifth of the patients had chronic pneumonitis, which appeared to be a consequence of repeated aspiration of regurgitated stomach contents.

A possible antecedent role of patulous cardia in the development of a hiatal hernia is discussed.

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DISCUSSION

(PAPERS BY OGURA [PAGE 340], AND HIEBERT [PAGE 352] AND THIER ASSOCIATES)

DR. IVAN A. MAY, Oakland, Calif.—I rise to suggest that left colon transplant may be more satisfactory than right and to present a series of 4 patients operated upon during the past year at Highland Hospital, using the left colon for total esophageal replacement. Prior to this the right colon was used in four instances. In the first case the colon was not long enough and it was necessary to complete the cervical end of the anastomosis by skin flaps using the technique described by Wookey. Subsequent dilatations of cervical stricture have been necessary. In the third case the blood supply was not adequate and resulted in a fistula. The right colon proved satisfactory in the other two, but was obviously inadequate in 2 of the 4 cases.

Dr. William Byrne, therefore, suggested that we use the left colon because of its longer length and more constant marginal artery. This we have done with excellent results. One may use only the transverse and descending colon or may use any amount of the sigmoid necessary to obtain adequate length.

The first operation was performed in October, 1960, in two stages. The entire sigmoid was used bringing it retrosternally to the neck. Seven inches of excess colon was left hanging out the cervical incision for 2 weeks before completing the operation—a rather unusual looking necktie.

Subsequently we have not used any sigmoid and have had no difficulty with length or blood supply. Primary anastomosis in the neck has been performed.

After transplant, barium swallow cinefluorography shows that these patients do not empty the colon as satisfactorily in the prone as in the upright position. The function is best when the colon lies in position to allow a straight line fall. We, therefore, place it behind the stomach loosely in the retrosternal space and excise excess colon from the upper end and *also* from the lower end. It is important to avoid a loop due to excess length at the lower end. This assures a straight line drop from the esophagocolic to the cologastric anastomosis.

[Slide] Here we see the transverse and descending colon lying on the chest, freed but for the mid-colic vessels. The upper end is above the mid-neck and demonstrates the more than adequate length and excellent color and blood supply.

DR. GEORGE H. A. CLOWES, JR., Cleveland, Ohio.—Recently, with the advent of cinefluorography for the study of esophageal function, we have applied this to colon replacements. It is of interest to find that after a year or more the transplanted colon appears to lose its peristalsis and dilates slightly. The barium refluxes upward, whether or not the colon is put in in an isoperistaltic fashion. We thought it was important, after

our early experiences, to put the colon in in an isoperistaltic fashion and I still believe we are having better results if we do so. I rather gathered that in Dr. May's cases, with the use of the left colon, the colon must be in the antiperistaltic direction, but this doesn't seem to matter, so far as the colon is concerned, in withstanding the acid secretions. Apparently, it is capable of protecting the remaining esophagus above it. I expect this is true also in Dr. Burford's patients, but I should be interested to know, when he has made these very high repairs of the type that he has described, whether the patients have acid reflux into the mouth through such a long antiperistaltic segment of colon.

DR. FREDERICK S. CROSS, Cleveland, Ohio.—We have had some experience with incompetency of the gastric cardia without x-ray evidence of a hiatal hernia, and reported on this experience several years ago at the Association Meeting in Los Angeles.

We have had a smaller, and I must admit less picturesque, series of 10 patients to date, all of whom have been markedly improved or completely cured of their symptoms of regurgitant esophagitis by reconstituting the cardiac sphincter mechanism, as in hiatal hernia repair.

[Slide] It is possible to demonstrate an associated hiatal hernia in about 75 to 80 per cent of the patients with esophagitis. If such a hernia is present most patients can be helped by repair of the hernia. On the other hand, 25 per cent of patients in whom no hernia is found present somewhat of a problem. In certain patients the esophagitis can be related to a specific precipitating cause, such as vomiting in pregnancy, the presence of a nasogastric tube, or food or alcoholic excess. This is usually an acute esophagitis that can be handled quite successfully with the usual medical means.

Finally, there are the remaining patients with esophagitis of long standing, resistant to medical therapy, in whom no hernia has been demonstrated by x-ray examination. How are they to be treated?

Before answering this question I would like to make some comments on the diagnosis of a hiatal hernia. With regard to the small hernias, I would like to re-emphasize the comments of the authors that these are often reported as "insignificant" hernias by the radiologist. They are not in the position to make the diagnosis of "a small insignificant hernia." The true significance of a hernia can be ascertained only on the basis of symptoms and by esophagoscopy to evaluate the degree of associated esophagitis.

[Slide] It is not uncommon to demonstrate a hiatal hernia only after repeated x-ray examinations, especially if the examinations are of the routine upper gastrointestinal type. This was true in about 6 per cent of our 103 patients with esophagitis. This slide demonstrates the barium swallow in a patient in whom no hernia was demonstrated by x-ray examination. She was treated unsuccessfully for 2 years for esophagitis by accepted medical means. A second study showed the hernia, which has been repaired, and she remains asymptomatic.

[Slide] This is the barium swallow from another patient which was reported negative for hernia. On a more careful study of the cardiac area I felt this was a significant roentgenogram in that there is evagination of the gastric mucous membrane above the esophagogastric junction. This will generally be reported as a negative finding; we think it is a significant change. More important, this patient had significant esophagitis by esophagoscopy, and was helped by repair of the hernia.

To get back to the original discussion and to answer the previously posed question; many patients with significant esophagitis and no hernia shown by routine x-ray studies will actually have a hiatal hernia that can be demonstrated by more precise studies, including cinefluorography and closer interpretation of films. If no hernia is seen, regurgitation of barium from the stomach into the esophagus may be shown.

These patients can be helped by reconstituting the cardiac sphincter mechanism as in hiatal hernia repair.

DR. ELLIOTT MICHELSON, Baltimore, Md.—I should like to discuss the incompetence of the gastric cardia without radiologic evidence of hiatus hernia. The authors

depend upon the history of postural aggravation of symptoms and esophagoscopy for the diagnosis of gastroesophageal reflux. A more objective method for determining reflux may be obtained by acid perfusion of the esophagus and manometric determination at the gastroesophageal junction. Drs. Mendeloff (Tr. A. A. Physicians, 1961), Hendricks, and Siegal have performed these tests on 25 consecutive patients with esophagitis and found that those with symptoms, when perfused with N/10 HCl, developed pain and showed abnormal spastic contractions of the lower esophagus. In all cases of esophageal reflux, there was decreased pressure at the gastroesophageal junction. In patients with patulous cardia but without symptoms, there was no reaction to acid perfusion and esophageal motility was not abnormal. In those patients in whom medical treatment had brought relief of symptoms, acid perfusion did not provoke symptoms, nor was esophageal motility markedly abnormal. In one patient with achlorhydria with esophageal symptoms, acid perfusion did not reproduce the symptoms but his own gastric juice provoked the reaction.

[Slide] The first slide depicts the normal positive pressure zone, with the peak on inspiration at the gastric cardia. When the gastroesophageal junction is reached, the pressures increase slightly and are biphasic on inspiration. Above this junction, the esophageal pressures become negative with the peaks in expiration. In the patulous cardia, with or without hiatus hernia, the stomach pressure remains the same, the biphasic segment at the juncture reveals the same pressure as the proximal esophageal area.

[Slide] In the normal esophagus, following a "wet" swallow, a progressive peristaltic wave is seen moving downward. With perfusion of N/10 HCl in the patient with esophagitis, the peristaltic wave is replaced by spastic contraction at the time pain is elicited.

Acid perfusion of the esophagus and manometric studies are excellent objective procedures in the diagnosis of esophagitis and gastroesophageal reflux.

DR. ARTHUR M. OLSEN, Rochester, Minn.—I enjoyed Dr. Hiebert's presentation very much. We have all been interested in the contributions that come from Mr. Belsey and his group and from all of our other colleagues in the British Isles who have contributed so much to our knowledge of the diseases of the esophagus.

Like the previous speaker, I should like to offer some physiologic evidence supporting Dr. Hiebert's findings. [Slide] We have been using a manometric technique of measuring pressures at the esophagogastric junction for a number of years. In normal individuals, an elevation of pressures can be demonstrated that corresponds to the lower sphincter of the esophagus. The studies of Dr. Ellis and my colleagues at the Mayo Clinic have indicated that this sphincter is probably the most important factor in preventing reflux into the esophagus. However, there are a certain number of apparently normal people [slide] who have a very weak lower esophageal sphincter. This finding is illustrated in the second slide, which shows no elevation of pressure at the esophagogastric junction. This patient suffered from "heartburn," especially at night.

This manometric technique is useful in the detection of small esophageal hiatal hernias which cannot be demonstrated at roentgenographic examination or by esophagoscopy. The motility findings of such a hiatal hernia are noted on the last slide. [Slide] This tracing shows that the lower sphincter of the esophagus is above the diaphragmatic hiatus. This particular patient had the clinical symptoms that one would expect with incompetence of the gastric cardia. We have found that the electromagnetic pressure transducer is a valuable aid in the diagnosis of hiatal hernias as well as other disturbances of the esophagus and in sphincters.

DR. DONALD B. EFFLER, Cleveland, Ohio.—My associate, Dr. Laurence K. Groves, and I have encountered the same problem and have made similar observations. Unfortunately, we have included these in our series of cases of hiatal hernias. Perhaps this has been a mistake on our part as there is growing evidence to suggest that significant incompetence at the gastric cardia can and does occur without associated hiatal hernia.

Apparently our thinking on this surgical problem coincides with that of Drs. Hiebert and Belsey. We have used a similar method of repair and our results in a smaller series of cases have been gratifying. I do not recall a single patient who has not had complete relief after this comparatively simple transthoracic repair.

It now seems apparent that patients with significant incompetence at the gastric cardia can obtain relief only by proper surgical repair. It has been my impression that many surgeons have become distracted by other surgical procedures that do not attack the problem head on. It is for this reason that many surgeons today advocate pyloroplasty, with or without vagotomy, for reflux esophagitis. It would seem that this is an indirect approach and of questionable value. Perhaps more serious is the tendency toward radical procedures utilizing interposition by jejunum or colon. It is our opinion that operations of this magnitude are rarely necessary, unless the diseased esophagus has suffered irreversible disease.

For those surgeons who have not utilized the simple operative procedure described by Drs. Hiebert and Belsey, I can only urge that they give it serious consideration.

DR. OGURA (Closing).—In answer to Dr. May's question about the left colon, I think that this can be used.

On Dr. Clowes' question about acid reflux, we have not observed gastric reflux into the colon. The operation has been deliberately staged for two reasons. Plastic repair of the hypopharynx requires a tracheostomy. The staged procedure avoids the danger of possible communication between the tracheostomy and an open mediastinum.

This operation has further applicability. We have used it for high fistulas with complete paralysis of both cords where the colon anastomosis has been made with the pharynx. Subsequent plastic procedures to the vocal cord have given reasonably good function of the voice and excellent function of deglutition.

Finally, a warning about supraglottic resections. One must be very careful about the arytenoid cartilages. Surgical exposure of these cartilages will cause fixation and loss of function and permanent tracheostomy.

We further believe that an anatomic area where heretofore frustrating problems have bothered us has now been overcome by this combined service cooperation.

DR. HIEBERT (Closing).—I would like to make two points: First, a prediction that we will shortly agree that the common denominator of all esophagitis is gastroesophageal reflux, whether it be occasioned by a hiatal hernia, a patulous cardia, or by surgery to the esophagogastric junction. We will, furthermore, discard the notion that drinking strong coffee, alcoholic beverages, or putting a Levine tube through the area has anything whatever to do with the pathogenesis of esophagitis.

The second point is that we have a considerable job of re-education of our colleagues in radiology and internal medicine. If a patient complains of the symptoms we have talked about and the roentgenogram is negative, both patient and doctor are impressed that nothing serious is wrong. Moreover, the attitude is widespread that any thoracotomy is a very major undertaking.

Some time ago at Massachusetts General Hospital I had a patient with a hiatal hernia and I recommended that she have it repaired. She asked me how dangerous it was and I said, "Not very." She insisted, "Well, how dangerous?" I told her, "I was talking to Dr. Sweet and he remarked he had 500 consecutive hiatal hernia operations without a death." That was a mistake, because shortly after that Dr. Sweet had done 501.

CLINICAL CONSIDERATIONS AND SURGICAL TREATMENT OF ANNULO-AORTIC ECTASIA

Report of Successful Operation

Paul R. Ellis, M.D., Denton A. Cooley, M.D., and Michael E. De Bakey, M.D., Houston, Texas

ALTHOUGH most aneurysms of the thoracic aorta result from atherosclerosis, syphilis, or trauma, some occur because of an inherent structural defect in the aortic wall.[8, 13, 14, 16, 19] Such lesions are frequently associated with skeletal and ophthalmic manifestations of Marfan's disease and are generally considered to be cardiovascular manifestations of this syndrome.[10, 11] In actual fact, the original description of the clinical syndrome by Marfan[12] in 1896 included only skeletal abnormalities consisting of elongation of extremities and deformities of the skull. Subsequently, the ophthalmic and cardiovascular manifestations of this syndrome have become well recognized. McKusick[10] recently compiled an excellent monograph largely devoted to Marfan's syndrome, which describes the numerous anomalies that may be present in these unfortunate and often pathetic patients.

The development of thoracic aortic aneurysms in association with Marfan's disease was first described by Baer, Taussig, and Oppenheimer[1] as recently as 1943. Microscopically the basic process is one of medial degeneration, previously described by Erdheim[5] as cystic medial necrosis. Grossly these lesions usually begin at the aortic valve annulus and terminate proximal to the origin of the innominate artery. In some instances the aortic lesion is more extensive and involves the remainder of the thoracic and abdominal aorta.

Several cases are reported in which the typical aortic lesion is present in patients having a family history of Marfan's disease but lacking the typical skeletal and ophthalmic findings. These have been described as the "forme fruste" or incomplete form of Marfan's disease.[7, 18] McKusick[10] implies that for a patient to be a true example of Marfan's syndrome there must be either a family history of the disease or the characteristic dislocation of the optic lens. We have recently encountered a patient who had the typical thoracic aortic lesion but lacked both of these two criteria for the diagnosis of Marfan's syndrome. This case emphasized to us the need for an anatomically descriptive

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term to apply to patients having fusiform aneurysms of the ascending aorta caused by degenerative changes in the aortic wall. Inasmuch as the annulus of the aortic valve is usually involved in these patients, the term "annulo-aortic ectasia" is suggested. This designation could conveniently be applied to all aneurysmal lesions beginning at the annulus and terminating proximal to the origin of the innominate artery, irrespective of the basic etiological process.

Most patients having annulo-aortic ectasia have an associated aortic insufficiency due to dilatation and distortion of the aortic valve annulus.[9, 13, 14, 19] Surgical treatment of this lesion has in the past been unsatisfactory because of the technical problem concerned with resection of this segment of aorta, control

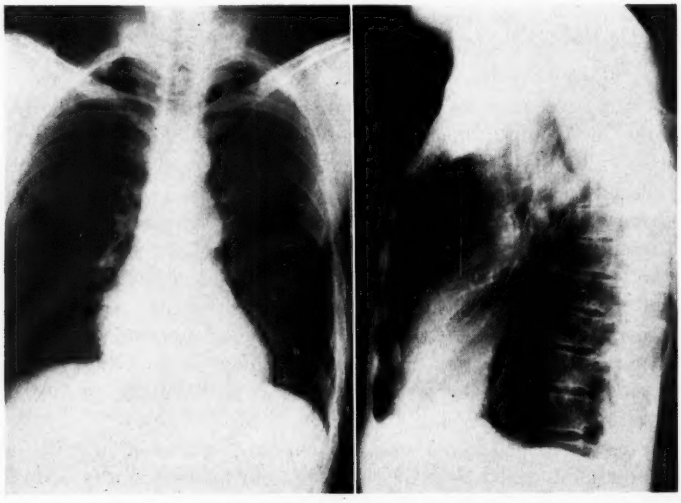

Fig. 1.—Roentgenogram of the chest made June 5, 1959, during first admission to hospital, demonstrating normal cardiac silhouette with mild pulmonary emphysema.

of the aortic regurgitation, and difficulty in obtaining a satisfactory permanent repair of the aorta by the usual technique of vascular anastomosis. In our patient having annulo-aortic ectasia with severe progressive aortic regurgitation, successful repair was obtained using temporary cardiopulmonary bypass. Clinical findings and the method of surgical treatment used form the basis for certain observations of practical importance made in this report.

CASE REPORT

C. G., a 45-year-old white man, was first admitted to Jefferson Davis Hospital on June 3, 1959, with complaints of weakness, dizziness, and shortness of breath of 3 days' duration. He denied any significant illness in the past. Family history revealed that both parents lived to an age of 65 or 70, dying of unknown causes. The patient had a brother who was 50 years of age and in good health.

Physical examination disclosed an acutely ill, mildly cyanotic patient who became dyspneic on slight exertion and upon sitting up. The extremities appeared normal in length and there were no skeletal deformities. Ophthalmoscopic examination revealed mild chorioretinitis without evidence of lens displacement. The blood pressure was 110/90 mm. Hg

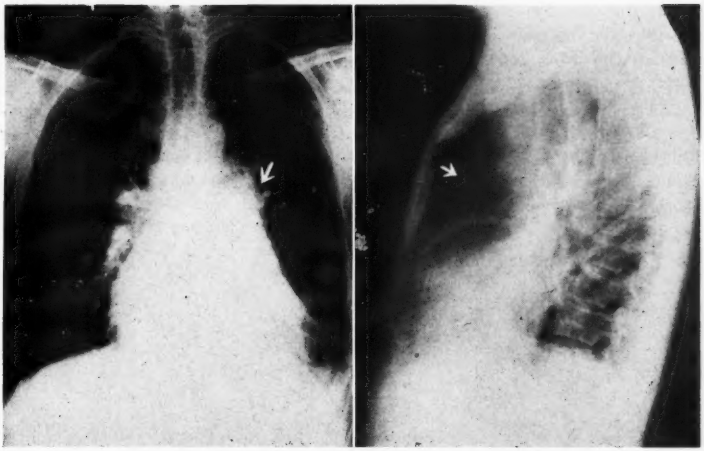

Fig. 2.—Roentgenogram of chest made Jan. 12, 1960, at the time of re-admission to hospital showing considerable increase in heart size since previous admission. Note left lateral and anterior displacement of pulmonary artery segment (*arrows*) by annulo-aortic lesion.

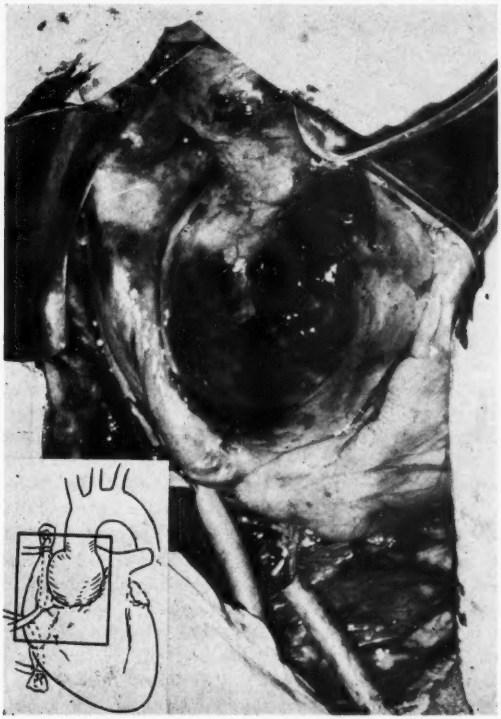

Fig. 3.—Photograph made during operation demonstrating fusiform aneurysm of annulo-aortic area. Inset identifies location of operative field which is visible.

and there were no cardiac murmurs. Serologic studies were negative for syphilis. Roentgenograms showed a normal cardiac silhouette with evidence of pulmonary emphysema (Fig. 1), and the electrocardiogram suggested right atrial enlargement.

Right heart catheterization was performed and revealed normal pressures in the right ventricle and pulmonary artery without evidence of an intracardiac shunt. Peripheral arterial oxygen saturation at rest was 94 per cent. Pulmonary function studies were performed and showed a ventilatory and diffusion defect compatible with mild pulmonary emphysema. The patient was discharged on June 19, 1959, with the diagnosis of pulmonary emphysema and fibrosis.

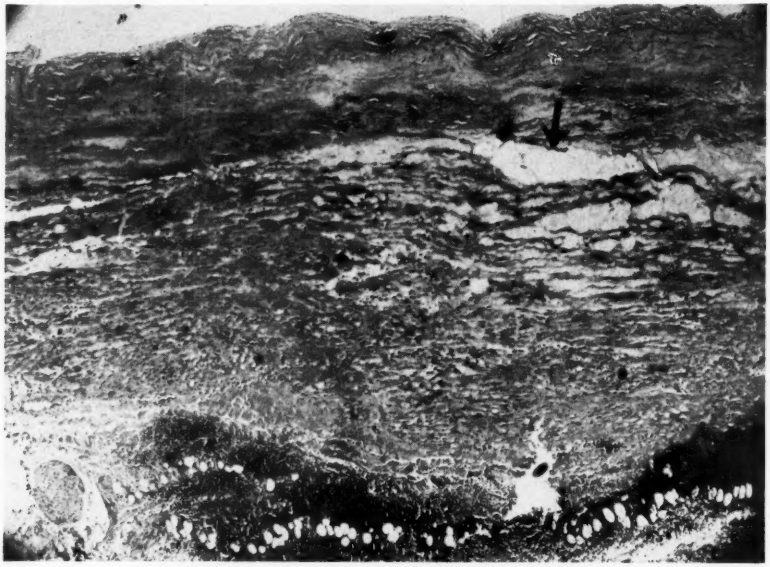

Fig. 4.—Photomicrograph of specimen of aortic wall showing degeneration with cystic changes in media and fragmentation of elastic fibers.

He was again admitted on Jan. 10, 1960, with ankle edema and progressive dyspnea of 3 weeks' duration. Physical examination revealed a Grade IV blowing diastolic murmur of maximum intensity along the left sternal border. The blood pressure was 130/50 mm. Hg, and water-hammer pulses were present. Roentgenograms demonstrated a marked increase in heart size with a prominent pulmonary artery segment (Fig. 2). In the lateral view there was obliteration of the retrosternal space which suggested right ventricular hypertrophy.

It was apparent that the patient had severe aortic insufficiency with congestive failure. In spite of intensive medical management his condition continued to deteriorate and operation was recommended. On March 22, 1960, the chest was opened through a median sternotomy incision. The sinuses of Valsalva and the ascending aorta were involved in an aneurysm approximately 6 cm. in diameter arising in the aortic annulus and extending into the ascending aorta, terminating about 4 cm. proximal to the origin of the innominate artery (Fig. 3). The pulmonary artery was of normal size but was displaced anteriorly and to the left. The right coronary artery arose from the lower third of the aneurysm anteriorly.

Preparations were made for total cardiopulmonary bypass using a Kay-Cross disc oxygenator and De Bakey roller pumps. After the bypass was instituted, the aneurysm was

incised vertically, the incision extending inferiorly toward the base of the noncoronary cusp. The vessel wall was very thin, but there was no evidence of a dissecting aneurysm. The valve leaflets were extremely large and thinned out, yet each was intact. Valvular incompetence was apparently due to the marked distortion of the annulus, probably permitting partial prolapse of the leaflets during diastole.

The repair began with bicuspidization of the aortic valve. The posterior or noncoronary cusp was excised along with the tremendously dilated sinus of Valsalva. The aortic aneurysm was then excised, particular care being taken not to disturb the ostia of the coronary arteries which were left attached to the aorta. Although an extensive aortic lesion was excised, direct suture of the proximal annulus and normal-sized ascending aorta was possible by end-to-end anastomosis.

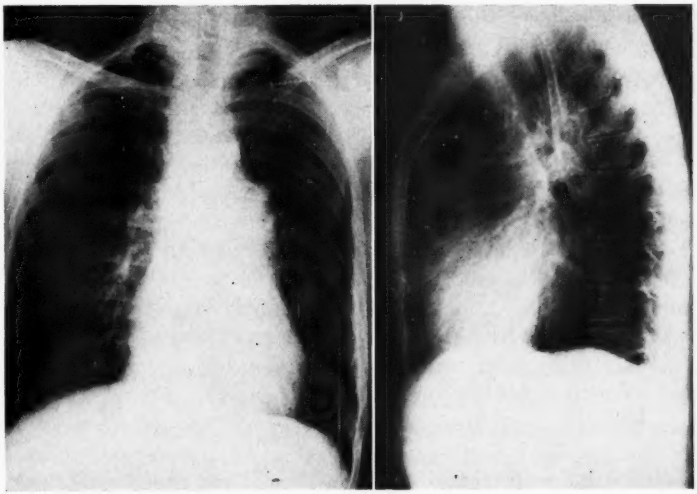

Fig. 5.—Roentgenogram made June 10, 1960, 3 months after completion of operation which shows persistent hilar enlargement on left although clinically the patient was asymptomatic and had no cardiac murmurs.

During the procedure the left coronary artery was intermittently perfused with oxygenated blood through a small polyethylene cannula from the pump oxygenator. The left side of the heart was decompressed with a metal suction tip inserted into the left ventricle through the aortic valve. Following restoration of circulation a tear in the thin-walled aorta was apparent along the posterior suture line, and it was necessary to reinstitute total cardiopulmonary bypass to repair the defect with additional sutures. Although the major bleeding was controlled, some ooze persisted at the suture lines which continued after closure of the chest, resulting later in an anterior mediastinal hematoma. The blood pressure remained stable, however, and re-exploration was not necessary. Microscopic sections of the aorta showed medial necrosis with degeneration of elastic fibers (Fig. 4).

After surgery the diastolic pressure remained between 60 and 70 mm. Hg and no diastolic murmur was audible. Pulmonary and peripheral edema disappeared and 4 months after operation the patient was doing well (Fig. 5).

DISCUSSION

The lesion of annulo-aortic ectasia is unusual in that it presents a double threat to the patient's life. First, the thin-walled aneurysm may rupture re-

sulting in fatal hemorrhage, and second, the presence of severe aortic insufficiency may cause intractable congestive failure, as in our patient. The clinical findings in advanced lesions consist of left- and right-sided failure occasionally associated with pain from the expanding vascular lesion. Physical examination reveals a diastolic murmur of aortic regurgitation associated with peripheral pulses characteristic of a wide pulse pressure. The lesion may not be detectable in the routine chest film, although anterior and left lateral displacement of the pulmonary artery segment should arouse suspicion. In those cases in which the diagnosis is uncertain, angiocardiography may demonstrate dilatation in the annulo-aortic region.[15, 17] Differentiation before surgery between this lesion and pure aortic valvular insufficiency is of importance to facilitate technical preparations for the more complicated procedure.

Successful surgical management of annulo-aortic ectasia depends upon the proper application of certain basic principles. First, the major portion of the wall of the aneurysm should be excised to remove the threat of possible rupture and fatal hemorrhage, and, second, the aortic valve incompetence must be corrected to restore adequate cardiac output. Early surgical efforts preceding the development of the pump oxygenator failed to accomplish these objectives and were unsuccessful. Bahnson and Nelson[2] performed aneurysmorrhaphy in 4 patients having an aneurysm of the ascending aorta. Two of these had associated aortic insufficiency and, although temporarily improved, both died within a year after surgery. Faye[6] attempted the same procedure in 1 patient without success. A Hufnagel valve was inserted by Dimond[4] to relieve the aortic insufficiency with subsequent failure and death.

Bahnson and Spencer[3] have recently reported their experience with aneurysms of the ascending aorta repaired during total cardiopulmonary bypass. Microscopically the wall of the aneurysm showed the degenerative changes of cystic medial necrosis. In several of these cases there was dilatation of the aortic annulus with aortic valve incompetence. The repair in these cases consisted of excision of the aneurysm and replacement with a prosthesis. An attempt was made to correct the aortic regurgitation by constricting the dilated annulus at the anastomosis to the prosthesis. Alhough this technique may be effective in those patients having mild regurgitation, we consider bicuspidization of the aortic valve a more effective procedure, especially in patients such as ours having severe dilatation of the annulus.

It is our opinion that annulo-aortic ectasia can be best corrected by utilizing an open cardiac procedure with total cardiopulmonary bypass. Coronary perfusion is necessary to prevent myocardial ischemia unless hypothermic arrest is employed. A method for decompression of the left side of the heart with return of bronchial artery blood to the system should also be provided. Usually this is best accomplished with a sump type of suction tip inserted in the left atrium. Furthermore, closely woven Teflon or Dacron prostheses of varying sizes should be available for replacement of a segment of ascending aorta in extensive lesions. This type of prosthesis is most suitable since leakage is minimal through the graft even in the presence of heparin.

During excision of the thin-walled ascending aortic aneurysm, special attention should be given to the attachment of the coronary arteries. These often originate from the wall of the aneurysm making it necessary to leave a small segment of the aorta with the coronary arteries attached. As previously mentioned, this lesion characteristically terminates below the origin of the innominate artery providing normal aorta above for restoration of continuity of the vessel.

The basic defect causing aortic insufficiency is dilatation of the aortic annulus, and this, we believe, is best corrected with bicuspidization of the aortic valve. The posterior or noncoronary cusp is partially excised and the commissures of the remaining two cusps are approximated. Bleeding will often occur from the suture line at the base of the aorta, and we recommend that the anastomosis in this area be reinforced with a second row of stitches, preferably of 4-0 Mersilene (Dacron) with small needles.

Surgical treatment of annulo-aortic ectasia should be reserved for those patients in whom the lesion is life threatening. These include patients showing progressive dilatation of the ascending aorta in whom rupture is a potential hazard and patients who have severe aortic regurgitation resulting in progressive congestive failure. In cases of early ectasia of the annulo-aortic area, operation should be withheld until progression of the disease can be demonstrated.

SUMMARY

Annulo-aortic ectasia is a term suggested for an aneurysmal dilatation of the ascending aorta and aortic annulus frequently associated with valvular regurgitation. The basic structural defect in the aortic wall may be cystic medial necrosis which may or may not be accompanied by stigmata of Marfan's syndrome.

Surgical treatment consisting of excision of the aneurysm and repair of the aortic regurgitation by bicuspidization of the valve was successful in the reported case. Temporary cardiopulmonary bypass with intermittent coronary perfusion is recommended for support of the patient during open repair of this complicated lesion.

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EXPERIMENTAL REPLACEMENT OF THE PULMONIC VALVE WITH A TRICUSPID SEMILUNAR PROSTHESIS

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NUMEROUS problems have been encountered in the construction and experimental implantation of valvular prostheses.[1-6] Among these are thrombosis, the design of a valve geometrically suitable for implantation into the heart, and the development of operative techniques which permit rapid insertion of the value with minimal insult to myocardial function. It would appear that the latter two problems have received inadequate attention. Accordingly, in the present studies a newly designed tricuspid prosthesis was evaluated by replacement of the pulmonic valve during right heart bypass. The method of construction of the valve, the technique for its fixation, and the hemodynamic results of its implantation are presented.

THE PROSTHETIC VALVE

The prosthesis employed is a tricuspid valve molded of polyurethane foam* (Fig. 1). The outer portion of the valve is 2.8 mm. thick and is of a compressible foam structure (Fig. 1, *C*). The leaflets, 0.15 mm. thick, provide minimal resistance to outflow. Compression is used during the molding process to avoid an open foam structure of the leaflets. The outer margin of the valve is designed to permit its attachment to the natural origin of the semilunar valves. Since slight twisting or compression of the valve results in sufficient distortion of the cusps to produce incompetence, a thin metal ring constructed of brass, or stainless steel, and having the same semilunar shape as the margin, is molded into the valve near the origin of the cusps (Fig. 1, *B*). The mold employed was constructed of metallized plastic (Devcon) which permitted inexpensive modification of the mold shape during development of the valve.

The configuration of the valve mimics the semilunar valves of the dog, both in the design of its cusps and their insertion into the walls of the pulmonary artery and aorta. In order to determine the optimum diameter for the

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*Stafoam, PE 102, American Latex Products Corp., Hawthorne, Calif.

prosthesis, the circumferences of the pulmonic and aortic valve rings were measured in fresh canine autopsy specimens. The calculated diameters of the pulmonic and aortic valves in these specimens are plotted in relation to body weight in Fig. 2. From these data, an outer diameter of 2.0 cm. was chosen for the prosthesis, whereas the metal supporting ring was made 1.5 cm. in diameter. Thus, the valve may be inserted into relatively large dogs and, by compression or trimming of the external portion of the valve, it may be inserted into smaller animals as well.

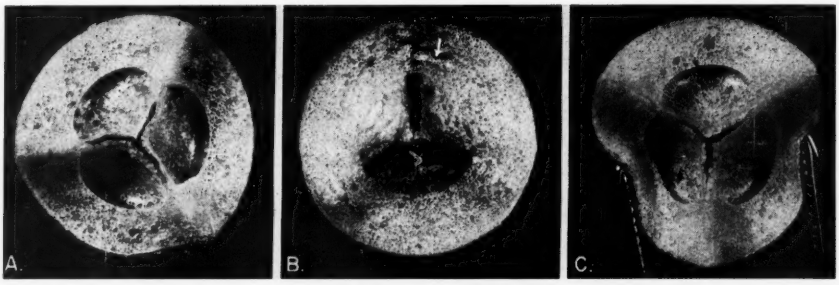

Fig. 1.—Photographs of the molded semilunar tricuspid prosthesis of polyurethane. *A*, Pulmonary artery surface. *B*, Right ventricular surface. *Arrow* indicates the partially visible segment of supporting metal ring. *C*, Compression of the outer portion of the prosthesis causes no significant distortion of the valve leaflets.

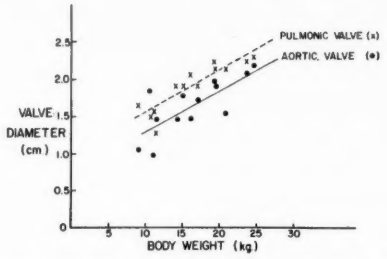

Fig. 2.—Relationship of body weight to the calculated diameters of the pulmonic and aortic valve rings in fresh canine autopsy specimens.

When water was pumped at pulsatile flows through the valve in an extracorporeal circuit, pressure gradients of 0, 2, 5, and 7.5 mm. Hg were observed at forward flows of 1,000, 1,800, 3,000, and 3,750 ml. per minute, respectively. The valve was competent with a static reverse pressure head in excess of 90 mm. Hg. The design of the cusps prevented their inversion and regurgitation occurred only with stretching or tearing of the leaflets.

METHODS

Fifteen dogs, weighing from 13.5 to 21.5 kilograms (average 16.2 kilograms) in body weight, were anesthetized with pentobarbital sodium; in 5 of these experiments, succinylcholine was also administered intermittently, and, in 4, 0.01 mg. per kilogram of atropine sulfate was injected preoperatively. In

9 dogs, Digoxin (0.02 to 0.025 mg. per kilogram) was administered prior to operation. Systemic pressures were obtained through catheters placed in the femoral artery and vein, and intracardiac pressures were measured by direct needle puncture of the pulmonary artery and right ventricle. The pressures were measured with Statham P23A transducers and recorded on a multichannel oscillograph.

After tracheal intubation, a left thoracotomy was performed through the fourth intercostal space, the pericardium was opened, and tapes were passed around the superior and inferior venae cavae, and the main pulmonary artery. The animal was given 2 mg. per kilogram of heparin and a cannula, 14 Fr., was placed into a segmental pulmonary artery for perfusion of the lungs.

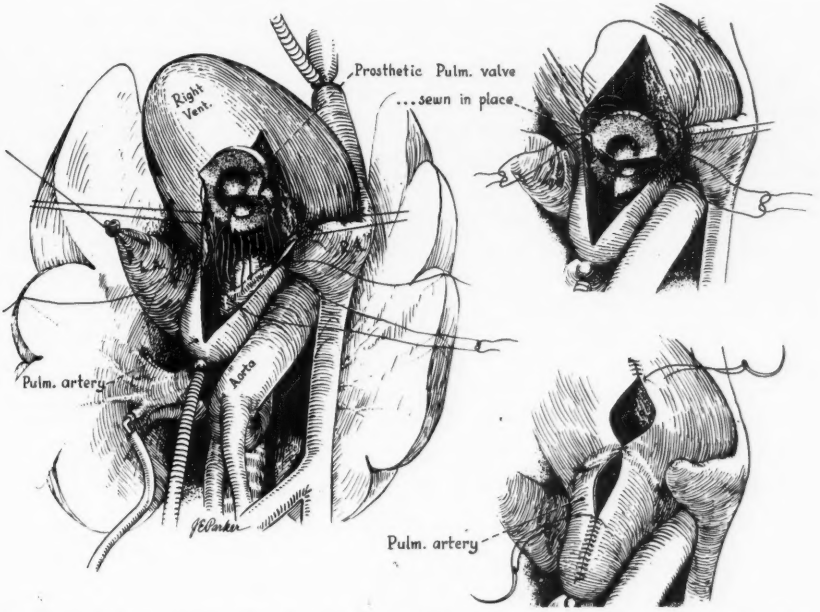

Fig. 3.—Technique of implantation of the prosthesis into the pulmonic valve annulus after institution of right heart bypass. See text.

Large bore cannulas were inserted through the femoral vein into the inferior vena cava. These venous cannulas drained by gravity into a reservoir from which a Sigmamotor pump provided the flow to the pulmonary arteries. Right heart bypass was initiated by occluding the venae cavae and the main pulmonary artery with tourniquets. A vertical incision was made into the pulmonary artery and extended downward through the anterior cusp of the pulmonic valve into the right ventricle. Coronary sinus and azygos vein flow were returned to the venous reservoir by a suction tip attached to a second Sigmamotor pump. The normal pulmonic valve was then excised. Implantation of the prosthesis was accomplished with three continuous mattress sutures

of braided 3-0 silk (Fig. 3). The first suture was placed at the origin of the right anterior commissure of the normal valve, and was passed through the pulmonary artery from the outside inward. It was then carried posteriorly as a continuous mattress suture through the margin of the artificial valve and along the remnant of the excised valve to the point of origin of the posterior commissure; the final bite of this suture was passed through the valve leaving the free end of the suture within the pulmonary artery. The second suture was initially passed through the valve at the site of termination of the first suture and then continued to the point of origin of the left anterior commissure where the free end was passed through the pulmonary artery. These two continuous mattress sutures were drawn up and tied together at the posterior commissure leaving the knot within the pulmonary artery. The third and final mattress suture was introduced through the wall of the pulmonary artery at the left anterior commissure and continued across to the right anterior commissure. The free margins of the pulmonary artery were then approximated, the third suture was tightened and its free ends were tied to the adjacent free ends of the first two sutures. A single reinforcing mattress suture was then placed anteriorly at the junction of the pulmonary artery and the right ventricle. The pulmonary artery was closed with a continuous everting suture of 5-0 braided silk, the ventriculotomy was approximated with a double row of silk sutures and normal circulation was reinstituted. Upon completion of the perfusion, either protamine sulfate or Polybrene was administered intravenously until coagulation in the wound was apparent; the dosage was usually two to three times that of the initial heparin dose.

RESULTS

The pressure measurements obtained at the operating table following insertion of the prosthetic valve are shown in Table I. The pressure gradients across the valve ranged from 0 to 25 mm. Hg, averaged 9.6 mm. Hg, and in all but 3 dogs were below 14 mm. Hg. The systolic pressures in the right ventricle were below 35 mm. Hg in 12 of the 15 dogs (Fig. 4). In each instance the diastolic pressure in the pulmonary artery was well maintained. Pressures in the femoral artery and in the inferior vena cava were within normal limits in all 15 animals.

In the first 6 dogs, the operative procedure was prolonged, since several methods of implantation were assayed. In the valves examined at autopsy 2 to 7 hours after insertion, little or no fibrin deposition on the valve cusps was observed. In the last 9 dogs, efforts were made to shorten the anesthesia and operative times and it was possible to insert the valve in a minimum of 23 minutes of perfusion time with a total operating time of 90 minutes. Tracheobronchial secretions were usually heavy, postoperative respiratory difficulty was prominent, and survival usually did not exceed 36 hours. For this reason, in 4 experiments atropine was administered preoperatively in an attempt to decrease secretions. There was one chronic survivor in the latter

group, and heavy secretions were not apparent in either of the 3 animals that died 1 to 2 days postoperatively. Of the eight valves examined at 1 to 2 days postoperatively, two showed moderate thrombus deposition and partial fusion of the valve commissures (Fig. 5), one showed heavy deposits of white thrombus, and one valve was heavily coated with almost complete obliteration of the commissures and partial fixation of the cusps (Fig. 5). These findings are summarized in Table I.

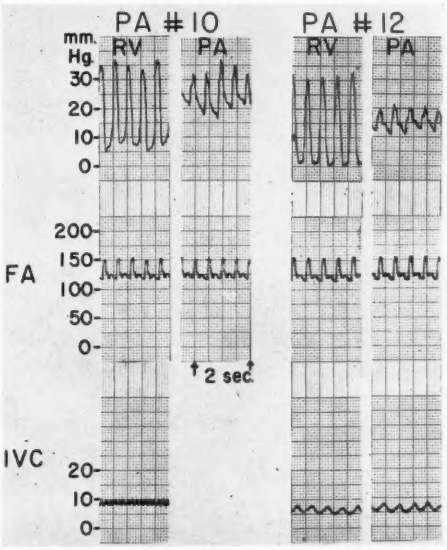

Fig. 4.—Representative pressure tracings obtained immediately following insertion of prosthetic valve. *RV* = right ventricle; *PA* = pulmonary artery; *FA* = femoral artery; *IVC* = inferior vena cava.

TABLE I. HEMODYNAMIC AND PATHOLOGIC OBSERVATIONS AFTER PULMONIC VALVE REPLACEMENT

DOG NO.	WEIGHT (KG.)	PRESSURE (MM. Hg)					SURVIVAL	AUTOPSY FINDINGS	EXTENT OF VALVE THROMBUS
		RV	PA	SYSTOLIC GRADIENT	IVC	FA			
1	14.0	20/3	20/15	0	—	93/70	Acute		Slight
2	20.0	29/5	18/13	11	9	107/70	Acute		Slight
3	19.8	Operative death							
4	19.2	38/3	25/12	13	4	130/95	Acute		None
5	21.5	30/3	18/12	12	5	130/108	Acute		Not examined
6	15.3	22/1	17/3	5	—	110/92	Acute		None
7	16.4	26/2	16/10	10	7	135/105	1½ da.	Atelectasis	Heavy
8	15.0	45/4	20/10	25	4	130/77	3 hr.	Hemothorax	None
9	17.9	30/1	25/14	5	6	117/92	12+ hr.	Atelectasis	None
10	16.0	33/9	33/18	0	—	150/115	8 hr.	Hemothorax, atelectasis	Heavy
11	15.0	23/3	11/6	12	4	163/118	16 hr.	Atelectasis	Slight
12	17.2	32/3	20/12	12	6	155/115	Chronic		
13	16.0	32/2	22/17	10	5	165/107	12+ hr.	Hemothorax	Moderate
14	14.7	42/0	22/14	20	5	155/95	1½ da.	Atelectasis	Moderate
15	13.5	33/2	23/17	10	5	152/107	12+ hr.	Hemothorax	Slight

RV = Right ventricle. PA = Pulmonary artery. IVC = Inferior vena cava. FA = Femoral artery.

Catheterization of the surviving dog, 1 month after operation, revealed a right ventricular pressure of 76/2 mm. Hg. The pulmonary artery was not entered. The right ventricular pressure obtained immediately after insertion of the prosthesis in this dog was 32/3 mm. Hg and the peak systolic gradient across the valve was 12 mm. Hg.

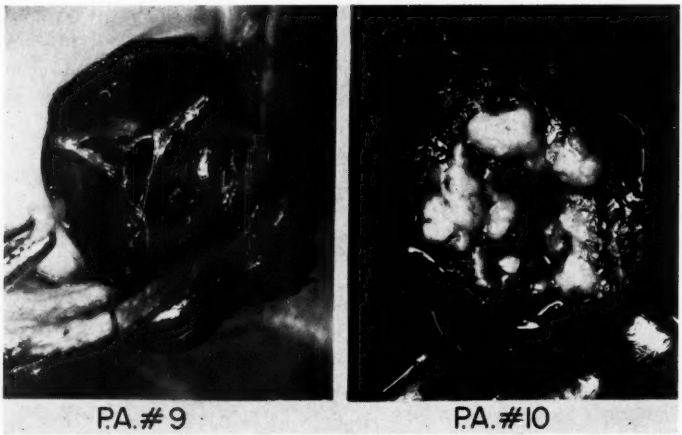

Fig. 5.—Photographs of the prosthetic valve at postmortem examination. PA 9 shows partial fusion of commissures, but no gross thrombus formation. PA 10 shows heavy white thrombus deposition.

DISCUSSION

The operative technique described is simple and provides a relatively rapid method for routinely obtaining secure fixation of the valve. In addition, it gives minimum intravascular exposure of suture material. The valve design utilized in these studies proved satisfactory from a hemodynamic standpoint, and its further experimental application appears warranted. The pressure gradients observed were usually small and the pressure pulse contours indicated that the valve was competent at diastolic pressures up to 18 mm. Hg. Since the in vitro studies showed pressure gradient across the valve at flow rates approximating the expected cardiac output for these dogs (that is, below 2,000 ml. per minute), it seems likely that the gradients observed resulted from some compression of the leaflets after insertion, which decreased their mobility. This conclusion is supported by the observation that the 2 largest gradients were measured in 2 of the smaller dogs. The outer compressible foam margin of the valve proved advantageous to its implantation in dogs of various weights. Since the aortic ring is only slightly smaller than the pulmonic ring (Fig. 2), it is anticipated that the valve should be suitable for implantation in the aortic area in dogs of approximately the same size as those used in these experiments.

The polyurethane foam used in the fabrication of the valve is fragile and may not be of sufficient strength or durability to withstand for an extended period the high diastolic pressure and the rapid velocity alterations encountered in the aortic area. Although this might be remedied by incorporating a strengthening fabric,[4] the frequency of heavy thrombotic deposition on urethane valves observed by us and by others[1, 7] suggests that the search should continue for materials with better structural qualities and a surface less prone to promote clotting. Braunwald,[4] however, in experiments with polyurethane mitral valves has found that clotting is not necessarily a problem with this material. Although solid silicone rubber has been promising,[6] it would appear advantageous to retain a foamed material for the valve margins, and to provide highly flexible and strong cusps of the same material, but without the foamed structure, since fibrin deposition within the foam might be expected to reduce the mobility of the leaflets.

It is recognized that the pulmonic valve is not essential to survival of the dog but replacement of this valve as an investigative technique has several advantages. Although the pressure stress is less than in the aortic area, blood flow through the pulmonic orifice is identical and pressure gradients developed across the valve should be comparable. In addition, the semilunar attachment of the pulmonic valve closely resembles that of the aortic valve, and the absence of coronary arteries facilitates the study of implantation methods and valve design. The use of right heart bypass also offers advantages, since the volume of blood required to prime the apparatus is small and the equipment is simple.

SUMMARY

A newly designed tricuspid semilunar valve prosthesis is described, an operative technique for its insertion into the pulmonary annulus is presented, and experiences with its insertion into 15 dogs are discussed. Immediate hemodynamic function of the valve was satisfactory although, in several animals, subsequent thrombotic deposition on the valve surface was observed. The advantages of pulmonic valve replacement as an investigative tool in the study of valve design and structure are discussed.

The aid of Messrs. Alexander G. McInnes, John M. Debroske, James J. Gilliam, and Harry J. Knight in the fabrication of the valve is gratefully acknowledged.

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SUPERIOR VENA CAVAL REPLACEMENT

V. Use of Fresh Homografts of Superior Vena Cava

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THE IMPORTANCE of obtaining a suitable and consistently successful means of superior vena caval replacement and bypass has been discussed in detail in previous communications.[3, 5, 6, 7] Many graft materials which have been successful in aortic and arterial replacement have been found to be unsatisfactory for vascular replacement in the low pressure venous system. The present study was carried out to evaluate the suitability of fresh homografts of superior vena cava for superior vena caval replacement in dogs and to study the histologic fate of fresh caval homografts in this low pressure system.

MATERIAL AND METHODS

Healthy mongrel dogs were used. The weight of the dogs generally was in the range of 8 to 12 kilograms. Intravenous Pentothal was utilized for anesthesia. Oxygen was administered through an endotracheal tube by means of a mechanical respirator. A right third intercostal space incision was employed.

When the right pleural cavity was opened, the superior vena cava was dissected free from surrounding structures. The pericardium was opened in the area of the entrance of the superior vena cava into the right atrium. A Potts coaretation clamp was placed across the right atrium caudad to the entrance of the superior vena cava into the right atrium and a Glover vascular clamp was employed to occlude the superior vena cava and its major branches just cephalad to the origin of these branches. A segment of superior vena cava, ranging from 2 to 3 cm. in length, was resected between the occluding clamps.

In the initial group of animals, the fresh caval grafts were obtained by two teams of surgeons working simultaneously at adjoining tables. The resected superior vena caval segment from one animal was promptly passed from one table to the other and was used to replace the resected superior vena caval segment in that animal. In this manner, superior vena caval replacement with a fresh homograft of superior vena cava was carried out simultaneously in 2 animals. This method of caval replacement was employed in the first 24 grafts studied (Dogs 928 through 795 in Table I). However, one of the caval grafts often proved to be shorter than the gap of superior vena cava to be spanned and varying degrees of tension with graft narrowing often occurred in these animals. In order

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TABLE I. SUMMARY OF EXPERIENCE WITH THE USE OF FRESH HOMOGRAFTS OF SUPERIOR VENA CAVA FOR SUPERIOR VENA CAVAL REPLACEMENT

	FIRST CAVOGRAM		SECOND CAVOGRAM		THIRD CAVOGRAM		FOURTH CAVOGRAM		POSTMORTEM FINDINGS			
DOG	DAYS AFTER OPERATION	PATENCY	DAYS AFTER OPERATION	PATENCY	DAYS AFTER OPERATION	PATENCY	DAYS AFTER OPERATION	PATENCY	DIED OR SACRIFICED	DAYS AFTER OPERATION	PATENCY	RESULT
928	42	Patent*							S	42	Patent*	Patent*
926									D	82	Occluded	Occluded
936									D	16	Occluded	Occluded
937	84	Occluded							S	84	Occluded	Occluded
938									D	2	Patent	Patent
939									D	26	Occluded	Occluded
941	75	Occluded							S	75	Occluded	Occluded
942	74	Patent*	153	Patent	399	Patent	462	Patent				Patent†
954	28	Patent	148	Patent	392	Patent			S	467	Patent	Patent
957	70	Patent*	148	Patent*					S	210	Patent*	Patent*
958									D	51	Patent	Patent
972									D	21	Patent	Patent
974									D	36	Patent	Patent
986									D	28	Occluded	Occluded
897									D	2	Patent	Patent
760									D	9	Patent	Patent
761	56	Patent	127	Patent	372	Patent	439	Patent				Patent†
762									D	9	Occluded	Occluded
764									S	129	Patent*	Patent*
770									D	2	Occluded	Occluded
773	42	Patent	113	Patent	357	Patent	421	Patent				Patent†
774									D	2	Patent	Patent
777	35	Patent	113	Patent					S	217	Patent	Patent
778	35	Patent	113	Patent								Patent‡
795	35	Patent	106	Patent					S	152	Patent	Patent
667									D	103	Patent	Patent
133	70	Patent*							S	174	Patent*	Patent*
139									D	55	Occluded	Occluded
173	63	Patent							S	169	Patent	Patent
174									D	63	Occluded	Occluded
194	56	Patent							S	162	Patent*	Patent*
198	56	Patent							S	184	Patent	Patent
213	49	Patent							S	153	Patent	Patent
219	49	Patent							S	153	Patent	Patent
259	14	Patent										Patent‡

*Evidence of stenosis or narrowing on cavogram or at autopsy.
†Living, with recent cavogram.
‡Lost from colony.

to overcome this problem, the last 11 grafts were obtained by resecting a relatively long superior vena caval segment from one animal and using it to replace a considerably shorter resected caval segment in the other. The long resected caval segment was replaced by a crimped vascular graft of Teflon.[4] In this manner, the fresh caval homograft could be inserted without tension on the graft and the resultant narrowing of the lumen.

The anastomoses were carried out with a continuous over-and-over suture of 6-0 arterial silk. Bleeding at the suture line was seldom a problem. The azygos vein was ligated in continuity in the majority of the animals after completion of the anastomoses and the release of the occluding clamps. No effort was made to cover the caval replacement with pleura. The thoracotomy incision was closed in layers and a catheter which was left in the chest during the closure was aspirated and withdrawn.

RESULTS

Superior vena caval replacement with fresh homografts of superior vena cava was carried out as described in 35 dogs. The patency of the graft was investigated by cavogram, autopsy, or both, from 2 to 467 days after operation (Table I). Twenty-five of the grafts remained patent (71 per cent) and 10 were occluded. Stenosis, varying from mild to severe in degree, was encountered in 5 of the patent grafts on postmortem examination. Three animals are living, 2 are missing from the colony, and 30 are dead. Fourteen animals were sacrificed and 16 died. In the group of animals that died, 8 of 16 grafts were patent. The graft was patent in 12 of the 14 sacrificed animals. The 2 occluded grafts in this group had been shown earlier to be occluded on cavogram.

In the group of animals in which 2 caval homografts were inserted simultaneously by transferring the resected caval segment from one animal to the other, 16 of 24 grafts remained patent (67 per cent). In the group in which a longer caval segment was obtained by replacing the caval segment of the donor animal with a Teflon graft, 9 of 11 grafts (82 per cent) remained patent.

Cavograms were of value in evaluating the patency of the homografts without sacrificing the animals and in following the continuing patency and functional adequacy of the grafts (Fig. 1). Eighteen of the animals were studied by cavogram. A second cavogram was obtained in 8 animals, a third in 4, and a fourth in 3. Three animals are living with a recent cavogram showing graft patency from 421 to 462 days after operation. A third cavogram in one animal showed graft patency without narrowing or stenosis 392 days after operation. When this animal was sacrificed 467 days after operation, the graft was found to be patent and functioning satisfactorily (Fig. 2).

The initial cavogram in the 18 animals was made from 14 to 84 days after operation. At the time of the initial cavogram, 16 grafts were found to be patent and 2 were occluded. Three of the patent grafts were found to have some narrowing or stenosis of the graft. However, all 3 grafts were still patent on subsequent autopsy and cavogram evaluation, 104, 140, and 388 days after the initial cavogram. One of these animals, Dog 942, was found to have some narrowing of the lumen of the graft on initial cavogram, 74 days after operation. Subsequent cavograms showed an increase in the caliber of the

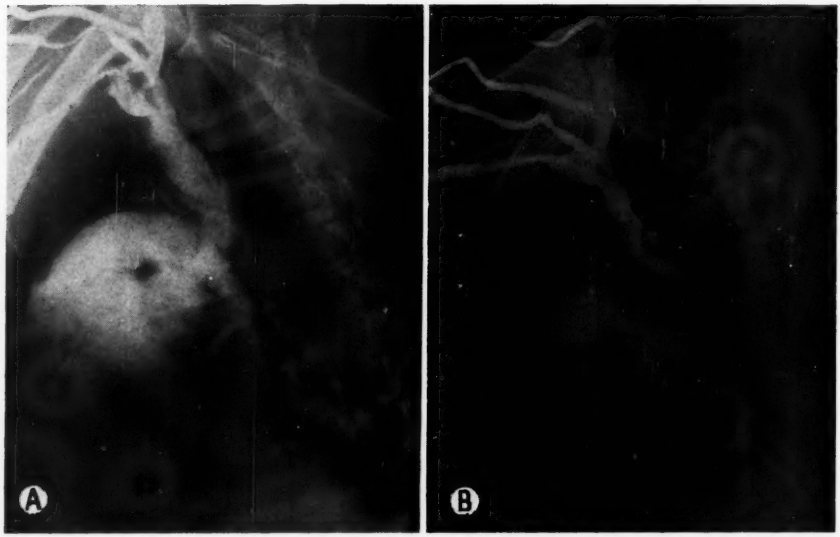

Fig. 1.—Cavograms in Dog 761 showing patency of homograft in superior vena cava 56 days after operation *(A)* and 439 days after operation *(B)*.

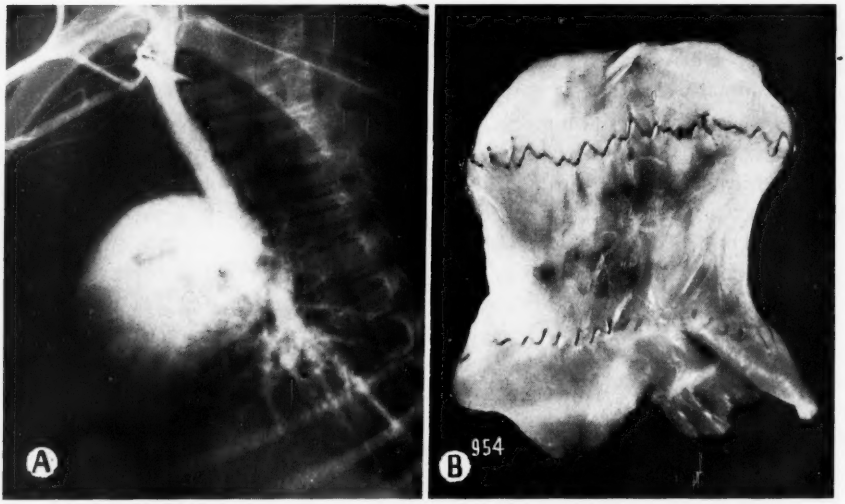

Fig. 2.—Cavogram appearance of homograft in superior vena cava of Dog 954, 392 days after operation *(A)* and postmortem appearance of the same homograft at sacrifice 467 days after operation *(B)*.

lumen and the most recent cavogram, made 388 days after the initial cavogram, showed a significant increase in the size of the lumen as compared with its appearance at the time of the initial cavogram (Fig. 3).

Excluding Dog 928, which was sacrificed on the day of the initial cavogram and Dog 259 which was lost from the colony after the initial cavogram, 14 animals in which graft patency was demonstrated at the time of the initial cavogram have been followed by subsequent cavograms, autopsy, or both, from 78 to 439 days after the initial cavogram. It is of interest that the homograft had remained patent in all 14. The interval from the initial cavogram to the most recent evaluation of graft patency has been in excess of 1 year in 4 of these animals.

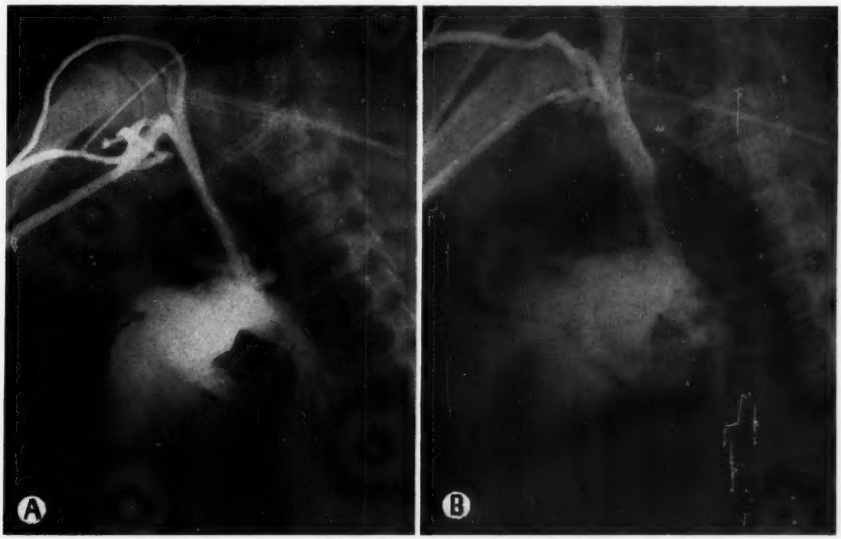

Fig. 3.—Cavogram appearance of homograft in Dog 942 *(A)* 74 and *(B)* 462 days after operation. There is some narrowing of the homograft lumen at the time of the initial cavogram. The most recent cavogram *(B)* shows a significant increase in the caliber of the lumen.

Thirty homografts were studied at the time of postmortem examination. Sixteen animals died and 14 were sacrificed from 2 to 467 days after operation. Twenty of these 30 homografts were patent. Some degree of stenosis was encountered in 5 of the patent grafts. Neither dilatation nor aneurysm formation were observed in any of the patent grafts (Fig. 4). When occlusion occurred, it appeared to be thrombotic in nature.

Ten of the caval homografts were subjected to careful histologic study from 51 to 467 days after operation. The specimens were fixed in 10 per cent formalin and longitudinal blocks were taken through the grafts. Microsections were made and stained with hemotoxylin and eosin, Masson's trichrome, and Verhoeff elastic stains.

Histologically, the normal canine vena cava resembles that of man. The intima consists of a single layer of endothelium which rests on a poorly defined

internal elastic membrane. Beneath the internal elastic membrane is a thin zone, the media. The media consists of a few collagen fibers and a few elastic fibers, both of which are loosely arranged. Ordinarily, smooth muscle is not seen in the media. The adventitia is the thickest of the three layers of the vena cava. It consists of smooth muscle, collagen fibers, and elastic fibers.

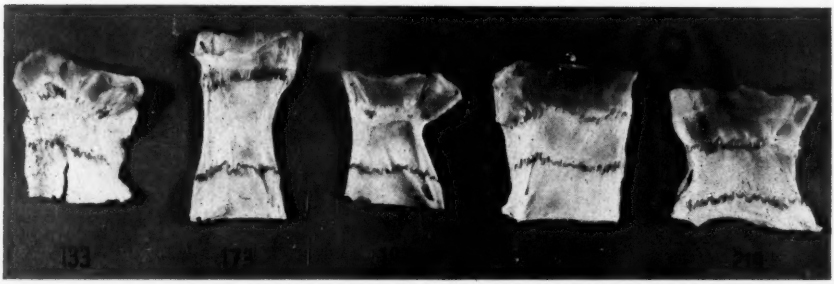

Fig. 4.—Photograph showing postmortem appearance of 5 patient hemografts at the time of sacrifice from 153 to 174 days after operation. A mild degree of graft stenosis had occurred in Dogs 133 and 194.

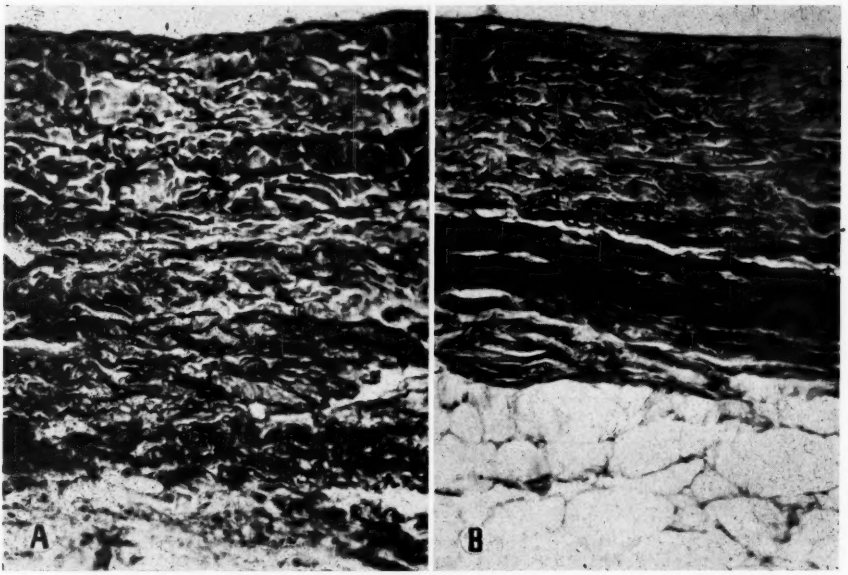

Fig. 5.—Photomicrographs with Verhoeff's elastic tissue stain showing the appearance of a normal canine superior vena cava *(A)* and the superior vena caval homograft of Dog 173, sacrificed 169 days after operation *(B)*. The elastic fibers appear as black lines. In the normal superior vena cava, the elastic fibers are separated by collagen fibers. In the homograft, a dark band of elastic fibers, in close proximity to one another, may be seen in the adventitia.

In general, similar histologic features were noted in all of the homografts studied (Fig. 5). The endothelium was intact in all. There was a limited amount of fibrous tissue beneath the endothelium. Occasionally, microscopic

foci of fibrous thickening of the intima were observed. In almost every instance, the internal elastic membrane was very poorly defined, fragmented, and discontinuous. The media of the homografts was thickened by fibrous connective tissue, so that its thickness equaled that of the adventitia. A few fragmented elastic fibers were observed in the media. The adventitia of the grafts contained an inner zone of many elastic fibers and an outer zone of fibrous connective tissue. In the inner portion of the adventitia, there were many layers of elastic fibers with very little fibrous tissue between the elastic fibers. The elastic fibers were thickened and fragmented and were located practically one on top of another. Elastic fibers were found in the oldest homograft studied, 467 days after operation. The elastic tissue, in all likelihood, represented the only part of the homograft to survive. Elastic tissue presumably does not regenerate. The intervening fibrous tissue that normally is seen between the elastic fibers of the vena cava presumably became necrotic and disappeared, allowing the remaining elastic fibers to come in close association with one another. The outer portion of the adventitia consisted of fibrous connective tissue. Smooth muscle was not observed in any of the grafts. Very little inflammation was encountered in the grafts and only a few scattered lymphocytes and plasma cells were seen in the adventitia.

DISCUSSION

Relatively little has been published concerning the use of venous homografts for superior vena caval replacement. Kay,[1] in a discussion of another paper in 1955, reported his disappointment with the use of venous homografts for superior vena caval replacement. He carried out caval replacement in 12 dogs.[2] Cavograms from 4 to 6 months after operation showed graft patency in approximately 30 per cent of the animals. However, by 6 months all of the grafts showed varying degrees of sclerosis and, by the time of sacrifice, none showed evidence that would permit him to describe it as a good graft.

The results of the study recorded here are somewhat more encouraging with respect to the suitability of fresh homografts of superior vena cava for superior vena caval replacement.

SUMMARY

1. The use of fresh homografts of superior vena cava for superior vena caval replacement in dogs is reported.

2. Thirty-five fresh caval homografts were used for superior vena caval replacement and the patency of the grafts was investigated by cavograms, autopsy, or both from 2 to 467 days after operation. Twenty-five of the homografts remained patent (71 per cent). Some degree of stenosis was encountered in 5 of the patent homografts. Three animals are living with patent homografts by cavogram from 421 to 462 days after operation.

3. Some degree of homograft tension was present in the initial group of 24 animals and 16 of the grafts remained patent (67 per cent). When stenosis

occurred in this group of animals it was relatively severe. There was no homograft tension in the last group of 11 animals and 9 grafts remained patent (82 per cent). Stenosis, when it occurred in this group, tended to be relatively mild in degree.

4. Cavograms proved to be of considerable value in assessing, at intervals, the patency of the homografts without sacrificing the animals. From 1 to 4 cavograms were made in 18 animals. Fourteen patent homografts were followed by repeated cavograms, autopsy, or both, from 78 to 439 days after the initial cavogram and all remained patent. Indeed, 1 animal which had some narrowing of the homograft lumen at the time of the initial cavogram was found on subsequent cavograms to have a progressive increase in the caliber of the graft lumen.

5. There was no evidence of graft dilatation or aneurysm formation in any of the patent homografts at the time of postmortem examination.

6. Ten homografts were subjected to careful histologic study by a variety of staining techniques from 51 to 467 days after operation. Only the elastic tissue of the homografts appeared to survive. The findings in all of the homografts studied were similar, suggesting that there was no progressive alteration or degeneration of the homografts after the 51-day interval after operation was passed. These findings give added support to the cavogram observations that fresh homografts which remain patent in the superior vena cava after an early critical period may be expected to remain patent.

7. The results of this study suggest that fresh homografts of superior vena cava may be used for superior vena caval replacement in dogs without tension with expectation of success in the neighborhood of 70 to 80 per cent and that the homografts which remain patent beyond an early critical period may be expected to retain their patency.

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HISTOLOGIC ASPECTS OF RADICAL CARDIO-OMENTOPEXY: VASCULARIZATION OF OMENTUM FROM THE AORTIC ROOT

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RADICAL cardio-omentopexy[2] was devised in our laboratories to overcome the effects of multiple blocks in diseased coronary arteries. The aim of the operation is to bring a new source of blood supply to multiple deep and superficial areas of the heart and to redistribute blood more equitably among various parts of the heart.

To do this, epicardium is removed chemically and surgically from the right and left ventricles. Multiple vascular strips of omentum are implanted into the left ventricle. After a light dusting of the denuded myocardium with talc, a wide strip of omentum is applied over the origin of the left coronary artery, over the conus arteriosus, and onto the origin of the right coronary artery. Remaining omentum is then applied to both right and left ventricles.

When radical cardio-omentopexy was compared with four other surgical procedures (Beck I, Vineberg sponge operation, ligation of the left pulmonary artery, and cardiac irradiation) for its ability to protect dogs in which the coronary arteries were gradually constricted,[3, 5, 6] only radical cardio-omentopexy provided statistically significant protection. Determination of myocardial blood flow[6] in the surviving dogs showed that several of the dogs had flow values approximating normal, despite the presence of constrictors on their coronary arteries.

We have been encouraged, as a result, to continue our exploration of various aspects of radical cardio-omentopexy. In this paper are presented histologic studies of the operation.

In all of our work with radical cardio-omentopexy, one of the most impressive features has been the ability of the omentum to parasitize multiple structures in the chest to bring extracardiac blood to the heart. Thus, in dogs whose left coronary arteries were occluded after performance of cardio-omentopexy, ligation of the omentum below the diaphragm 6 to 12 weeks after performance of omentopexy produced no clinical or electrocardiographic abnormalities.[2] At re-operation through the chest, however, when multiple adhesions of

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omentum to chest wall, pericardium, and lung were torn down, ligation of omentum close to the heart resulted in progressive electrocardiographic abnormalities and death.

This work raised the question of whether the omentum might be used to parasitize the root of the aorta itself to gain its new blood supply. It was hoped that by contact with the root of the aorta, laid bare by surgical and chemical methods, the omentum could draw a new blood supply directly from the root of the aorta, lead this supply into the heart, and, in effect, create multiple new coronary arteries.

METHODS

Cardio-omentopexy was performed in 24 normal dogs with implantation of 5 to 15 strips of omentum into the left ventricle. Histologic studies were carried out by injecting India ink and barium sulfate suspensions through the splenic artery into the omentum 5 to 90 days after the omentopexy. No constrictors were present on the coronary arteries of these dogs.

At the time of histologic study, a laparotomy was performed on each animal; a No. 20 polyethylene cannula was threaded into the distal splenic artery and the proximal end of the splenic artery was tied off. The dogs were heparinized (4 mg. per kilogram) and then killed by an overdose of Nembutal. All injections of contrast media were then made into the splenic artery distal to the ligature on the artery and below the diaphragm after death of the dog.

The vascular bed of the splenic artery was first flushed with saline solution while constant suction was maintained at the coronary ostia through an incision in the aorta. India ink (diluted with two volumes of saline solution) was then injected into the distal splenic artery. When carbon was seen to emerge from the coronary ostia, a 10 per cent suspension of barium sulfate in commercial 6 per cent dextran in saline solution was injected into the distal splenic artery until the barium sulfate could be seen emerging from the coronary ostia.

Best results with the barium sulfate were obtained when the suspension was made by violent agitation in a colloid blender, followed by passage through a standard intravenous administration set and, finally, violent blending again just before injection into the artery.

For histologic studies bearing on the ability of the omentum to gain a new blood supply directly off the root of the aorta, 3 normal dogs were prepared by bringing omentum through the diaphragm as before. The pericardial sac was opened anterior to the phrenic nerve. Visceral and parietal pericardium were otherwise completely undisturbed in order to prevent adhesions of omentum to the heart and pericardium. The periaortic fat pads were dissected off anteriorly, in each case producing enough bleeding to require fine chromic ligatures. The aorta was dissected free of the pulmonary artery. The posterior aspect of the aorta was scarified by the method of Beck.[1] For this, a gauze sponge wet lightly with a fresh solution of five per cent trichloroacetic acid in saline solution was pulled posterior to

the aorta and immediately removed. The omentum was drawn around the base of the aorta to cover the entire circumference of the denuded aorta. The omentum was sutured back on itself to maintain its position. The pericardial sac was then closed loosely.

Fig. 1.

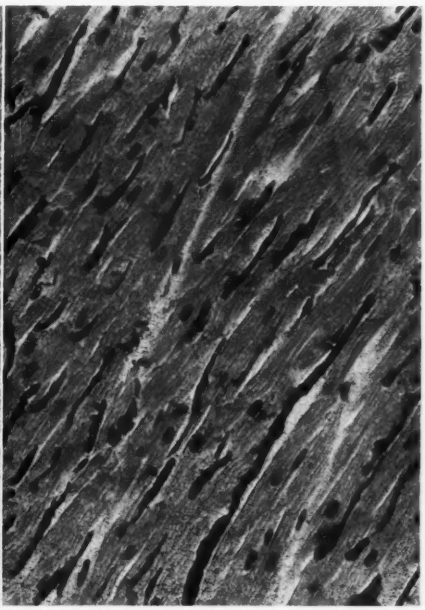

Fig. 2.

Fig. 1.—Microscopic section of heart of normal dog re-operated upon at 1 month after cardio-omentopexy. An omental implant is seen in the lower portion of the section. Two larger arteries *(arrows)* and multiple small ones in the omental implant contain barium sulfate suspension. The barium sulfate appears gray in contrast to the India ink seen in an artery in the myocardium *(upper left)* and in multiple smaller vessels throughout myocardium.

Fig. 2.—Slide from same anical shown in Fig. 1. , The India ink injected into the distal splenic artery below the diaphragm has traversed omentum to reach capillaries between muscle fibers of the heart.

Six months later the dogs were re-operated upon through a second thoracotomy incision. The omentum was immediately severed just above the diaphragm and the inferior portion of the omentum was tied off. The portion of the omentum attached to the aorta was left free and watched for viability and bleeding during the rest of the dissection. This portion of the omentum was then drawn cephalad and freed from adhesions to the lung and chest wall. The pericardial sac was opened through the previous incision. As noted earlier[4] because epicardium and parietal pericardium were not removed, omentum could be swept free of the heart and pericardium by simple digital pressure. By this maneuver the omentum was finally left supported only by its adhesions around the entire circumference of the aorta at the aortic root.

A No. 16 cannula was threaded into the arch of the aorta from the subclavian artery; the dog was heparinized and then killed by an overdose of

Nembutal. A Satinsky clamp was applied across the aorta just distal to the coronary arteries so that no medium could reach the omentum from the coronary vessels. The distal arch of the aorta was also clamped off. Contrast medium was injected through the subclavian artery into the proximal portion of the arch of the aorta.

India ink and barium sulfate suspensions, similar to those previously used, were injected until they could be seen emerging from the vessels of the omentum.

Fig. 3.

Fig. 4.

Fig. 3.—Cross section through omentum and aorta at the root of the aorta. Under high power magnification, the vessel marked with an *arrow* could be seen to contain barium. Carbon and barium sulfate could be traced from the vasa vasorum of the aorta into the omental vessels.

Fig. 4.—Cross section of omentum several inches from its attachment to the root of the aorta. Under high power magnification, the artery marked by an *arrow* could be seen to contain barium.

RESULTS

Five days after cardio-omentopexy in a normal dog, injection of the omentum through the splenic artery produced only carbon but no barium sulfate in the heart. In the remaining 23 dogs, studied 2 to 30 weeks after original omentopexy, both carbon and barium sulfate passed from splenic artery into the omentum and emerged from the coronary ostia by retrograde flow through the coronary arteries without difficulty.

Fig. 1 is a section of the heart of a normal dog studied 1 month after a cardio-omentopexy. All implants in this dog and the other dogs studied were

viable and contained one or more arteries carrying barium sulfate suspension. Carbon and barium sulfate could be seen in arteries within the heart in these dogs despite the fact that their coronary arteries had not been constricted.

Fig. 2 is a high power view of the heart of the same dog, in which carbon black is seen in capillaries between myocardial fibers.

In dogs in which the omentum was attached only at the root of the aorta, the omentum was watched for 30 to 60 minutes for signs of ischemia before the dog was killed and injection studies begun. In all cases, the omentum remained viable with no change in its color. Multiple hemorrhagic areas appeared at the cut surface of the omentum. Carbon and barium sulfate passed readily from the root of the aorta into the omentum.

Fig. 3 is a section of omentum attached to aorta. The vasa vasorum of the aorta as well as multiple omental arteries contained carbon and barium sulfate. Carbon and barium sulfate could also be seen in arteries of the omentum in sections taken several inches away from the aorta (Fig. 4).

The only difficulty in these experiments was in selecting portions of omentum containing small enough amounts of carbon and barium sulfate to allow microscopic sections to be cut without tearing the omentum. In most sections too much carbon and barium sulfate were present to allow good sections to be cut.

SUMMARY AND CONCLUSIONS

Histologic studies of cardio-omentopexy in dogs confirm the physiologic studies already obtained which show that omentum can supply blood to the ischemic heart. Vessels large enough to carry barium sulfate suspensions connect omentum and heart after cardio-omentopexy even to the hearts of normal dogs with open coronary arteries.

Just as omentum can gain a new blood supply from chest wall, pericardial sac, and lung, it can also gain a new blood supply directly from the root of the aorta.

If radical cardio-omentopexy is carried out for the clinical treatment of coronary artery disease, application of the omentum to the circumference of the denuded aorta just at the root of the aorta may be of value.

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THE OXYGEN CONSUMPTION IN MAN UNDER DEEP HYPOTHERMIA AND THE SAFE PERIOD OF CIRCULATORY ARREST

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THE REPORTED USE of deep hypothermia and circulatory arrest for open-heart surgery[7] raises an important question: Is it possible to predict what is a safe period of circulatory occlusion at a given body temperature from the values of oxygen consumption at that temperature?

This paper presents data on oxygen consumption obtained in cardiac patients operated upon in deep hypothermia and discusses these data in relation to the duration of circulatory arrest and the presence or absence of postoperative brain damage.

COOLING AND PERFUSION TECHNIQUE

A. *Complete Circulatory Arrest During the Period of Open-Heart Surgery.*—

The method described by Drew and co-workers[8] was used in the first 10 cases (Fig. 1; Table I). Blood was first drained from the left atrium at a rate of up to 10 ml. per kilogram of body weight per minute and pumped through a heat exchanger where it was cooled to 3° to 10° C. before entering the femoral artery. When ventricular fibrillation developed, blood was also drained from the right atrium or the caval veins and injected into the pulmonary artery by means of another pump. Total bypass continued with two pumps at a rate of 30 to 55 ml. per kilogram of body weight per minute, until the desired esophageal temperature was reached. In the first series the perfusion was completely stopped during the entire period of intracardiac surgery. After cardiac repair, total bypass with rewarming was started until the heart began beating. This occurred spontaneously in half the cases and after electric shock at 30° C. in the remainder. Thereafter, a partial perfusion was continued from the left atrium to the femoral artery until rewarming was satisfactory.

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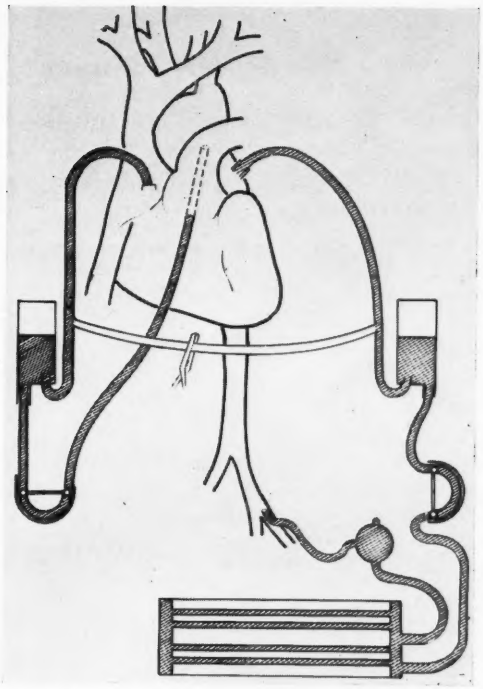

Fig. 1.—Perfusion technique according to Drew.[8] See text.

TABLE I. PROFOUND HYPOTHERMIA WITH COMPLETE CIRCULATORY ARREST

CASE NO.	AGE (YEARS)	LOWEST ESOPHAGEAL TEMPERATURE (° C.)	CIRCULATORY ARREST (MIN.)	DIAGNOSIS	COMPLICATION
1	26	15	25	Infundibular stenosis	0
2	32	7.5	47.5	Infundibular stenosis Pulmonary stenosis Atrial septal defect	0
3	19	10	21	Atrial septal defect	0
4	9	10	38	Totally abnormal venous return	0
5	12	10	44	Ventricular septal defect Infundibular stenosis	0 (Block)
6	18	10	27	Atrioventriculare commune	0
7	4	5	70	Tetralogy of Fallot	Brain damage, died after 102 da.
8	6	10	50	Tetralogy of Fallot	Brain damage, died after 47 da.
9	4	10	41	Atrioventriculare commune	Brain damage, died after 26 da.
10	11	8	49	Infundibular stenosis Pulmonary stenosis Ventricular septal defect Abnormal venous return Peripheral pulmonary stenosis	Inoperable, died on the table

B. *Low Perfusion Rates Without Oxygenator During the Period of Open-Heart Surgery.*—

In our next 7 cases (Table II) the technique just described was modified in order to allow perfusion without the oxygenator during the period of intracardiac surgery (Fig. 2). The coronary and pulmonary vascular beds were not perfused during this period.

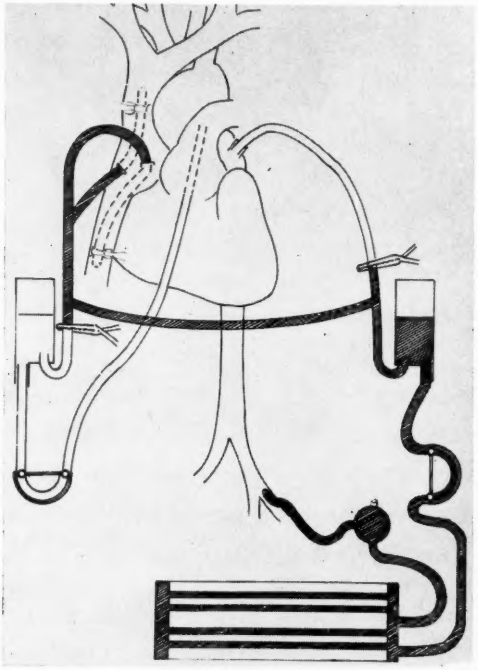

Fig. 2.—Modified technique with a slow perfusion with oxygen saturated blood from the venae cavae being pumped to the femoral artery during the cardiac arrest.

TABLE II. PROFOUND HYPOTHERMIA WITH LOW RATE OF PERFUSION

CASE NO.	AGE (YEARS)	LOWEST ESOPHAGEAL TEMPERATURE (° C.)	ARREST OF CORONARY CIRCULATION (MIN.)	DIAGNOSIS	COMPLICATION
11	17	10	41	Atrioventriculare commune	0
12	35	13	38	Atrial septal defect	0
13	29	11	40	Mitral insufficiency Mitral stenosis	0
14	3	11	38	Atrioventriculare commune	Block, died after 12 hr.
15	12	10	44	Ventricular septal defect	Inoperable, died after 5 da.
16	45	11	31	Aortic stenosis (calcified)	Inoperable, died after 12 hr.
17	29	11	70	Aortic stenosis (calcified) Aortic insufficiency	Inoperable, died on the table

TEMPERATURE RECORDING

Thermistors were inserted into the esophagus down to the level of the left atrium and about 15 cm. into the rectum. In addition, thermistor needles were inserted deep into the temporal muscle and into the arterial line of the extracorporeal circuit.

METHODS FOR DETERMINATION OF OXYGEN CONSUMPTION

During total body perfusion at a known rate of flow, samples of mixed venous and arterial blood were taken and analyzed for oxygen content according to Van Slyke and O'Neill. Oxygen consumption was calculated using the Fick principle, the blood minute volume and the arteriovenous oxygen difference being known.

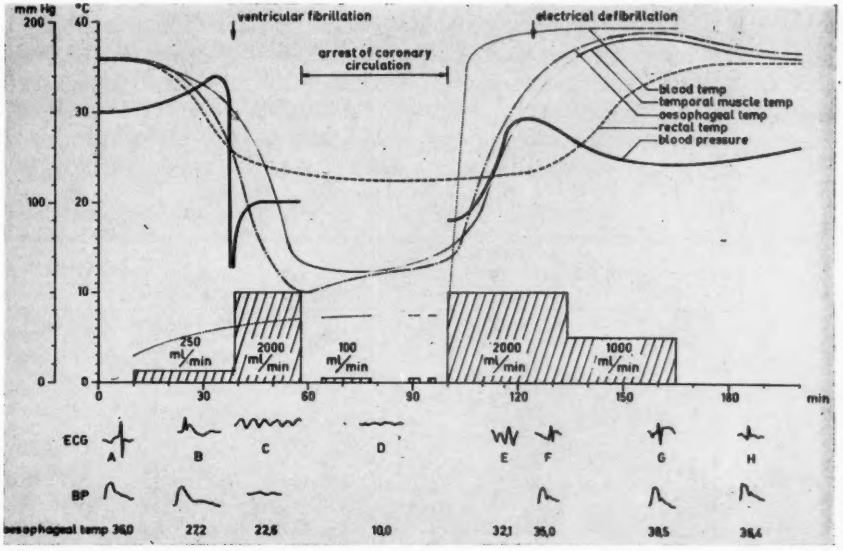

Fig. 3.—Case 11. Profound hypothermia with low rate of perfusion. Temperature and blood pressure (BP) diagram from a 17-year-old boy with A-V commune, who was perfused (method as in Fig. 2) under a cardiac arrest of 41 minutes and a slow perfusion during 24 minutes. Recovery was uneventful.

RESULTS

Rate of Cooling.—Table III shows the rate of cooling and the temperatures registered immediately before and at the end of the period of circulatory arrest. When the cooled blood was introduced into the femoral artery, the rectal temperature always fell faster than the esophageal until ventricular fibrillation started and total bypass began. The more rapid fall in esophageal temperature as compared with rectal temperature during total perfusion reflects the difference in blood flow to the various structures and the existence of temperature gradients. The temperature recorded by the needle deeply placed in the temporal muscle closely agreed with the brain temperature in dog experiments and

TABLE III*

CASE NO.	COOLING TIME TO LOWEST TEMP. (MIN.)	TEMPERATURE AT START OF OPEN-HEART PERIOD			TEMPERATURE AT END OF OPEN-HEART PERIOD		
		ESOPH. (°C.)	RECTAL (°C.)	TEMPORAL MUSCLE (°C.)	ESOPH. (°C.)	RECTAL (°C.)	TEMPORAL MUSCLE (°C.)
1	30	14.6	16.4	—	15.4	19.2	—
2	48	7.6	19.0	—	9.6	20.4	—
3	31	10.2	20.0	—	15.2	22.4	—
4	30	10.0	19.0	—	14.0	21.0	—
5	24	11.0	22.2	—	14.6	25.0	—
6	30	10.4	19.4	—	11.6	21.4	—
7	20	6.6	16.4	—	13.0	20.0	—
8	35	10.0	12.4	13.0	13.2	20.2	12.8
9	20	10.2	20.8	—	17.6	22.8	—
10	36	7.8	20.0	—	13.3	22.2	—
11	48	10.2	23.0	12.4	13.4	22.4	14.0
12	43	13.0	20.0	12.6	14.6	19.4	15.8
13	50	11.0	24.9	11.0	11.4	23.4	10.2
14	50	11.0	21.0	10.4	12.4	18.8	9.8
15	64	9.8	18.4	9.8	12.0	19.0	10.0
16	43	11.0	16.6	16.4	13.0	17.8	16.2
17	54	12.0	15.4	16.6	11.0	16.6	13.0

*Cases 1-10: complete circulatory arrest during open-heart period. Cases 11-17: slow rate perfusion of cooled blood during open-heart period.

TABLE IV. OXYGEN CONSUMPTION

AGE	BODY WEIGHT (KG.)	CASE NO.	SURFACE AREA* ($M.^2$)	BMR (O_2ML./M.)	TEMPERATURE °C.	
					ESOPH.	RECT.
12	26.5	5	1.01	164	18	24
					11	22
					10	22
18	49.9	6	1.54	197	19	21
					15	20
					12	19
					10	19
6	19.0	8	0.74	129	12	13
					10	12
4	15.4	9	0.66	101†	13	22
					11	21
17	57.0	11	1.72	263	20	25
					15	24
					10	23
35	40.3	12	1.38	170	15	23
					13	20
3	14.6	14	0.62	97†	12	21
45	72.4	16	1.88	242	15	22
					11	17
29	71.1	17	1.86	252	15	18
					12	15

*Surface areas by DuBois nomogram.
†From weight according to Benedict and Talbot.

usually corresponded well with the esophageal temperature (Table III). Because of the body temperature gradients, the esophageal temperature rose markedly during the period of circulatory arrest. This rise was partly counteracted in the last 7 cases by the maintenance of a slow perfusion rate of 2 to 6 ml. per kilogram per minute with cooled (5° to 10° C.) blood (Table III).

Oxygen Consumption.—Oxygen consumption rate was measured in 9 patients during total body perfusion (Table IV). The mean oxygen consumption rate at an esophageal temperature of 10° to 13° C. was 4 per cent (range 1 to 11 per cent) of basal metabolism rate (BMR), calculated according to the Mayo Foundation standards from the body surface.[6]

At an esophageal temperature of 18° to 20° C., the oxygen consumptions in 3 determinations were 16, 20, and 39 per cent (average, 25 per cent) of BMR. Determinations at 15° to 16° C. in 5 patients showed a mean oxygen uptake of 19 per cent of BMR (range 4 to 34 per cent).

In 5 cases (Table V) oxygen consumption was 2.7 per cent of BMR (range 1.2 to 4.2 per cent) at 10° to 13° C. esophageal temperature when there was a low flow perfusion with drainage from the caval veins being returned to the femoral artery. Even though there was no oxygenator in the system, the oxygen content in the "arterial" blood coming from the heat exchanger was still about 10 vol. per cent after low flow perfusion for 30 minutes.

RATE IN 9 PATIENTS

VOL. PER CENT O_2 CONTENT		FLOW	ML. O_2 UPTAKE/MIN. AT ESOPHAGUS (°C.)			O_2 UPTAKE IN % OF BMR AT ESOPHAGUS (°C.)		
ART.	VEN.	(ML./MIN.)	10°-13°	15°-16°	18°-20°	10°-13°	15°-16°	18°-20°
18.10	15.20	1,100			32			20
18.45	17.95	1,100	6			4		
18.60	18.45	1,200	2			1		
21.10	19.80	2,500			32			16
21.60	21.30	2,500		8			4	
21.80	21.70	2,500	3			2		
22.15	22.10	2,500	1			1		
17.90	17.10	850	7			5		
19.80	19.70	800	1			1		
17.55	16.80	750	6			6		
18.90	18.50	750	3			3		
16.30	11.10	2,000			104			39
17.70	13.15	2,000		91			34	
18.20	17.35	2,000	17			6		
15.80	14.70	2,000		22			13	
15.45	15.15	2,000	6			4		
15.30	14.55	750	6			6		
16.20	14.85	3,000		41			17	
17.45	16.95	3,000	15			6		
17.65	14.10	2,000		71			28	
18.35	16.95	2,000	28			11		
						Mean 4	19	25
						n 13	n 5	n 3

TABLE V

CASE NO.	MINUTES ON PERFUSION WITHOUT OXYGENATION	TEMPERATURE (°C.)		VOL. PER CENT O_2 CONTENT		FLOW (ML./MIN.)	O_2 UPTAKE (ML./MIN.)	O_2 UPTAKE % OF BMR
		ESOPH.	RECT.	ART.	VEN.			
11	15	13	22	13.85	7.25	100	6.5	2.5
12	12	12	20	15.15	12.30	200	5.7	3.4
	20	13	19	11.55	10.10	200	2.9	1.7
	28	14	19	11.10	10.10	200	2.0	1.2
14	29	11	19	9.60	7.55	200	4.1	4.2
16	27	13	18	12.25	9.95	250	5.8	2.4
17	30	11	16	8.70	6.30	400	9.6	3.8
	60	11	16	6.15	4.80	400	5.4	2.1
							Mean	2.7
							n	8

Duration of Complete Circulatory Arrest.—The duration of complete circulatory arrest varied from 21 to 70 minutes (Table I). Six of the 10 patients survived without postoperative neurological complications; the longest period of arrest in this group being 47 minutes (Case 2, Table I). Physiologic and psychologic tests of cerebral function were carried out in 4 of them, before and after operation.[23]

Of the 4 patients who died, 1 proved to be inoperable, while the other 3 survived operation and did not show signs of severe neurological complications immediately after operation. After 1 day, however, evidence of brain damage developed, such as ataxia, tremor, disturbance of balance, difficulty in swallowing, and talking. Two patients were extremely cyanotic with tetralogy of Fallot and 1 of them had a hematocrit of 82 per cent. The fatal outcome of the fourth case (No. 9) which occurred after 41 minutes of circulatory arrest is probably not related to the pronounced rise in temperature during the arrest (Table III).

In 7 cases, the method for maintaining a low perfusion rate during the period of open-heart surgery was used. Three patients survived without neurological sequelae, while 4 died (Table II). Case 14 recovered consciousness without apparent brain damage, but suddenly died of total heart block. In this case, however, postmortem microscopic examination showed evidence of brain damage,[21] even though perfusion continued at a low rate during the 38-minute period of open-heart surgery (Table V). There were 2 cases of aortic disease which could not be corrected adequately and the patients did not recover consciousness.

DISCUSSION

Bigelow and his co-workers,[3] using surface cooling in dogs, showed that, until cardiac arrest occurs between 18° and 20° C., the relationship between the fall in body temperature and the reduction in oxygen consumption was

almost linear. Extrapolation of the curve, they stated, would suggest that at body temperatures around 10° to 12° C. the oxygen consumption would be minimal, if not absent. However, Gollan,[11] in experiments with a pump oxygenator, found that at 10° C. there was an oxygen consumption of around 10 per cent, and of 5 per cent at 5° C. of control values (under anesthesia). Sealy and associates[18] used deep hypothermia combined with extracorporeal circulation in man. They also noted that the change in oxygen consumption per degree change in temperature was smaller below than above 20° C. These later reports confirm the work of Horvath[12] and Spurr[19] and their associates who showed exponential relationship between per cent of oxygen uptake and temperature.

Unequal cooling of various regions of the body makes it difficult to relate values on oxygen consumption rate to specific temperatures, such as esophageal, rectal, etc. However, when the esophageal temperature reached 10° to 13° C. and remained at this range before the period of open-heart surgery, it was believed that a relatively stable state was reached. Under these conditions we are more confident in stating the relationship between body temperature and oxygen consumption in our series at 10° to 13° C. esophageal temperature, an average of 3 to 4 per cent (maximum, 11 per cent) of basal oxygen consumption.

Uneven body perfusion, creating an oxygen debt, may have given false information about the metabolic rate, but this is unlikely as we did not encounter any metabolic acidosis before the period of circulatory arrest. Since there is no evidence of deficient oxygen utilization during hypothermia,[1] our interpretation is that the metabolism is slowed down to 5 to 10 per cent of normal at 10° C.

The investigation by Rosomoff[17] makes it probable that the decrease of brain metabolism during hypothermia parallels that of the total body. Since the human brain can tolerate 10 minutes of circulatory arrest at 30° C. body temperature (Waddell,[20] Björk[22] and others) when the metabolism is reduced by about 50 per cent, it should tolerate 50 minutes of circulatory arrest at 10° C. when the BMR is reduced by at least 90 per cent. This assumption is supported by the results of Niazi and Lewis[15, 16] who showed that monkeys survived up to 2 hours of circulatory arrest at body temperatures below 10° C. and that a patient tolerated 1 hour of circulatory arrest at 9° C. esophageal temperature.

Our results seem to indicate that other factors besides the metabolic rate may also determine the safe period of circulatory arrest. For example, in 1 case, although there was a 93 per cent reduction of BMR (No. 8, Table IV) before arrest and the esophageal temperature rose only 1.2° C. over the 50-minute period of circulatory arrest, nevertheless, fatal brain damage occurred. The estimated metabolic rate may have been inaccurate, because of uneven body perfusion. However, we had evidence of brain damage at microscopic examination in 1 case (No. 14) even with low flow perfusion and some oxygen supplied during the whole 50-minute period of open-heart surgery at 10° to 12° C. esophageal temperature. It is also of interest to point out that only children

suffered brain damage. The neurological symptoms did not appear immediately after operation and did not, as one would expect in general anoxia, predominantly point to damage of the brain cortex.

Factors other than general anoxia, such as the direct effect of cold and the sequestration of blood cells in the capillaries, may primarily be responsible for the observed brain damage. Jensen and colleagues[13] observed signs of severe neurological damage, predominantly motor ataxia, in dogs cooled to a brain temperature of 8° to 12° C. without occlusion of circulation. Dogs cooled to 12° to 18° C. did not show similar symptoms. On the other hand, it has been repeatedly demonstrated by other authors (Andjus,[2] Gollan[11] and their coworkers) that the body temperature of experimental mammals can be reduced to 0° to 1° C. with subsequent resuscitation and long-time survival.

In their studies on hypothermia to 20° C., Gelin and Löfström[9] observed intravascular aggregation of red blood cells and "white emboli" in the peripheral circulation. These emboli were tentatively ascribed to aggregation of white blood cells and thrombocytes and/or of intravascular clotting. Heparin did not completely prevent the appearance of a granular flow in their experiments. Infusions of low molecular dextran, however, prevented or reversed intravascular aggregation in induced hypothermia.[14] Our patients were given heparin, 5 mg. per kilogram of body weight. Low molecular dextran in a dose of 300 ml. in 4 cases and 500 ml. in 2 cases did not prevent brain damage in 2 patients (1 of them received 2.5 grams low molecular dextran per kilogram of body weight). It is possible that intravascular aggregation and capillary thrombosis were the primary causes of brain damage in some of our patients. We now use a higher flow when extracorporeal circulation is combined with deep hypothermia and also have an oxygenator in the circuit. Furthermore, we now use deep hypothermia only in adults to protect the myocardium against anoxia in surgery of coronary arteries, aortic valves, and aneurysm of the root of the aorta.[22]

SUMMARY

In deep hypothermia of 10° to 13° C., oxygen consumption of 4 per cent (range, 1-11) of the normal was found. The brain damage encountered in children during circulatory arrest is therefore not considered primarily anoxic, but probably is due to intravascular aggregation and capillary occlusion.

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THE SURGICAL TREATMENT OF PULMONARY COCCIDIOIDOMYCOSIS

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PULMONARY COCCIDIOIDOMYCOSIS continues unabated as a severe medical and surgical problem. Unlike tuberculosis, it has not been possible to reduce the incidence of the disease since the reservoir of infection is in the soil and small animals of the southwestern United States, rather than in infected human beings. Accordingly, any person living in, or traveling through this region, is liable to infection. The peripatetic nature of a portion of our populace, the location of military bases in this region, and the proximity of the region to vacation resorts and population centers have resulted in an increasing incidence of coccidioidomycosis.

In most instances, infection with *Coccidioides immitis* results in a mild upper respiratory tract infection of short duration with no significant sequelae. About 0.25 per cent of infected individuals will develop a progressive disseminating form of the disease with a mortality of 50 per cent or more. Approximately 5 per cent of patients with coccidioidal infection develop permanent pulmonary changes demonstrable roentgenographically.

During the period 1947 to 1957, 100 patients with pulmonary coccidioidomycosis were operated upon at the Fitzsimons Army Hospital. In each case the diagnosis was confirmed by identification of the organism in the sputum or in the resected specimen. Analysis of these cases has shown that residual pulmonary coccidioidomycosis often causes significant symptomatology. Furthermore, serious complications may follow resectional surgery. A new drug, Amphotericin B, administered intravenously, has been useful in combating these complications. The following report is an analysis of our experience with the surgical treatment of the pulmonary manifestations of this disease.

All patients in this series were military personnel or their dependents. Some of these cases have been described in reports by Forsee[1] and by O'Leary.[2] In the first 5 years, 23 patients were operated upon, whereas 77 patients have

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had operations for pulmonary coccidioidomycosis during the last 5 years. Eighty-six of the patients were men. All patients from whom a geographic history was obtained had resided in or passed through areas known to be endemic for this disease. Forty-eight patients had solitary granulomas, 48 air-containing cavities, and 4 patients presented with spontaneous pyopneumothorax (Table I). Fifty-seven per cent of the lesions were in the right lung and 43 per cent in the left. Sixty-nine per cent were located in an upper lobe. At present, 2 of the patients in this series are dead. Ninety-six per cent have been followed for from 2 to 10 years.

TABLE I. LABORATORY DATA IN 100 CASES OF PULMONARY COCCIDIOIDOMYCOSIS

	SPUTUM CULTURE	COCCIDIOIDIN SKIN TEST	COMPLEMENT FIXATION	SPECIMEN CULTURE
Cavitary lesions				
Positive	18	31	10	36
Negative	18	16	23	3
Not done	12	1	15	9
Solitary granulomas				
Positive	0	34	2	14
Negative	24	9	22	21
Not done	24	5	24	13
Pyopneumothorax				
Positive	4	2	3	2
Negative	0	1	0	0
Not done	0	1	1	2

The majority of individuals with solitary granulomas were asymptomatic except for occasional vague chest pain. The lesion was usually detected on routine roentgenography. Twenty per cent of these patients gave a history of severe upper respiratory infection within the past 5 years. In a few patients, chest films taken during that illness revealed pneumonitis in areas where the granuloma subsequently appeared. Characteristically the lesions were discrete and well circumscribed, although some had irregular margins. Calcification was observed in only 2 cases, and in 1 of these the nodule was enlarging. The granulomas ranged in size from 1.5 cm. to 3.0 cm. in diameter. Sputum cultures for fungi were performed in 24 instances with negative results in each case. Intradermal coccidioidin skin test 1:100 was done on 23 patients and was positive in 79 per cent. Complement fixation tests were positive in 2 of 24 cases examined. These solitary lesions were removed primarily for diagnostic reasons.

In the 48 cavitary cases, 50 per cent were asymptomatic. Sixteen of the patients gave a history of having had blood-streaked sputum or frank hemoptysis. In no patient was hemoptysis a primary indication for resection, although 2 patients had repeated hemoptysis. Among the symptomatic group, the majority was afflicted with a productive cough, fever, malaise, and weight loss. Sputum culture was positive for *C. immitis* in 50 per cent of 38 patients examined. The coccidioidin skin test was positive in 65 per cent of the 48 patients. Complement fixation test was positive in 10 of 33 patients examined. One patient

had a complement fixation titer of 1:80, the highest titer encountered, without clinical evidence of dissemination. The cavities ranged in size from 1 to 6 cm. in diameter.

It has been our policy to resect undiagnosed cavitary lesions for therapeutic and diagnostic reasons, even in the absence of symptoms. In addition, peripheral cavities and those over 4 cm. in diameter have been resected because of the danger of secondary infection or cavitary rupture. Of the 18 patients in whom the preoperative diagnosis of coccidioidomycosis had been confirmed by positive sputum culture, 12 had either severe symptoms, local spread, or enlargement of the lesion as visualized by x-ray examination. Six resections were done in patients with positive sputum and without significant symptomatology or change in x-ray studies. Three of these had cavities larger than 4 cm. in diameter and 1 had multiple cavities.

Four patients presented with pyopneumothorax, infected with *Coccidioides immitis* and presumably secondary to rupture of a coccidioidal granuloma or cavity. In 1 case, a solitary lesion had been observed 4 years previously. A second patient was noted to have had a small cavity shortly before the appearance of a pyopneumothorax. Coccidioidin skin tests were positive in the 2 patients tested. Culture of the pleural fluid was positive for *C. immitis* in all patients as was sputum culture in the 3 patients tested. These patients were moderately ill, febrile, and had a productive cough.

Three patients in the series had co-existent pulmonary tuberculosis. Their sputum cultures were repeatedly positive for acid-fast bacilli. In 2 of these, after antituberculous chemotherapy, bacteriologic examination of the resected specimen revealed only *C. immitis*. In the third patient, both *Mycobacterium tuberculosis* and *C. immitis* were found. A number of cavitary and solid lesions of coccidioidomycosis presented a clinical picture characteristic of pulmonary tuberculosis but without positive sputum. In these, a course of antituberculous chemotherapy had been administered prior to resectional surgery.

At operation, the coccidioidal lesions were frequently located peripherally, the cavity or granuloma being subpleural, with adhesions and pleural thickening marking the site. The granulomatous lesions often had a soft or liquid center. This indicated either liquefaction in the center of a caseous lesion or inspissation of a cavitary lesion whose bronchocavitary junction had been occluded. Evacuation of this material resulted in the appearance of a cavity. Calcification was uncommon in these lesions. Satellite lesions were less commonly seen than with histoplasmosis. The cavitary lesions usually had a moderately thick wall with satellite lesions in the surrounding lung parenchyma. By contrast, the x-ray appearance frequently suggested a thin-walled cavity. Examination of the cavity contents revealed cultures positive for *C. immitis* in 36 of 39 examinations. Cultures from the solitary granulomatous lesions were positive in 14 of 35 instances. Microscopic examination of the specimens revealed caseating granulomas not unlike tuberculosis. Special stains such as the periodic-acid Schiff or silver methanamine stains were used to demonstrate the organism

in the tissues. Small additional granulomatous lesions were frequently found in the bronchi adjacent to the resected specimens.

In the group of solitary granulomas a wedge resection was performed in 40 of 48 patients. In 4 of these, a partial decortication was also carried out. Segmental resection was performed in 7 and lobectomy in 1 case. For the cavitary lesions, 21 lobectomies were done with one concomitant decortication. Thirteen segmental resections and 13 wedge resections were also carried out. One patient underwent lobectomy combined with segmental resection. The 4 patients with pyopneumothorax had decortication combined with wedge resection in 3, and lobectomy in 1 case.

There were no operative deaths in the series. In the group of solitary granulomas, 1 patient developed cardiac arrest during induction of anesthesia, was resuscitated, and underwent resection of the lesion at a later date. Of the 4 patients with pyopneumothorax treated by decortication and concomitant resection, 1 patient developed a bronchopleural fistula after wedge resection. This closed after a thoracoplasty. The patient died 9 years later from coronary artery disease, without demonstrating evidence of recurrent coccidioidomycosis. In the cavitary group, 7 patients had postoperative complications with infection by *C. immitis.* Two patients developed bronchopleural fistula, sputum positive for *C. immitis,* and empyema from which *C. immitis* was grown. After thoracoplasty both of these patients had fistula closure and sputum conversion. Five patients developed recurrent cavitation in an area adjacent to the previous resection. In 4 of these, the sputum was positive. The fifth had a small cavity which closed spontaneously after 2 months of observation. In these 7 complicated cases only 1 patient had lobectomy; the remainder had either wedge or segmental resection (segmental resection was combined with lobectomy in 1 case).

The patients with recurrent cavitation have had severe symptoms marked by weight loss, productive cough, low-grade fever, and general malaise. Reoperation has been technically difficult and postoperative complications frequent. In our experience, the hilar dissection has been particularly difficult and hazardous. The postoperative complications of bronchopleural fistula and cavitary recurrence are reminiscent of the sequelae of pulmonary resection for tuberculosis, prior to the advent of antituberculous chemotherapy.

Until recently the chemotherapy of coccidioidomycosis has been inefficacious. In vitro, Amphotericin B was found to be fungicidal. Parenteral administration to animals was effective in controlling coccidioidomycosis and a variety of other fungal diseases. However, its clinical use was limited to oral administration with poor and irregular absorption without the desired results. Then a preparation was developed for intravenous use. The daily dose of 1 mg. per kilogram of body weight is dissolved in a glucose solution and given over a period of hours. It is necessary to gradually build up to this dose over a period of a week to minimize side reactions. Reactions have included severe thrombophlebitis, nausea, vomiting, fever, and temporary azotemia. However, sputum conversion has been accomplished after surgical failure.

CASE REPORTS

CASE 1.—An 18-year-old Caucasian soldier was transferred to this hospital from Texas, where a routine chest film revealed a 3 cm. cavity in the left upper lobe (Fig. 1, *A*). He was asymptomatic. The coccidioidin skin test was positive. Sputum cultures were repeatedly positive for *C. immitis*. In March, 1951, the lingula and anterior segment of the left upper lobe were removed for a 2.5 cm. cavity in the lingula. Satellite granulomas were present throughout the specimen, and cultures revealed *C. immitis*. The postoperative course was febrile, and films revealed multilocular cavities in the left upper chest. In October, 1951, he returned to duty, asymptomatic, and sputum negative. Three months later the patient developed cough and pain in the left upper chest. Chest films again revealed several cavitary lesions in the left upper lobe (Fig. 1, *B*). Complement fixation titer for Coccidioides was 1:80. In March, 1952, the apicoposterior segment of the left upper lobe was resected. There was marked technical difficulty due to fibrosis and friability of the major vascular structures. Because of a persistent postoperative space, a 4-rib thoracoplasty was carried out 2 weeks later. Postoperatively a bronchopleural fistula developed. After tube drainage the fistula closed. Five years after operation he is asymptomatic and gainfully employed.

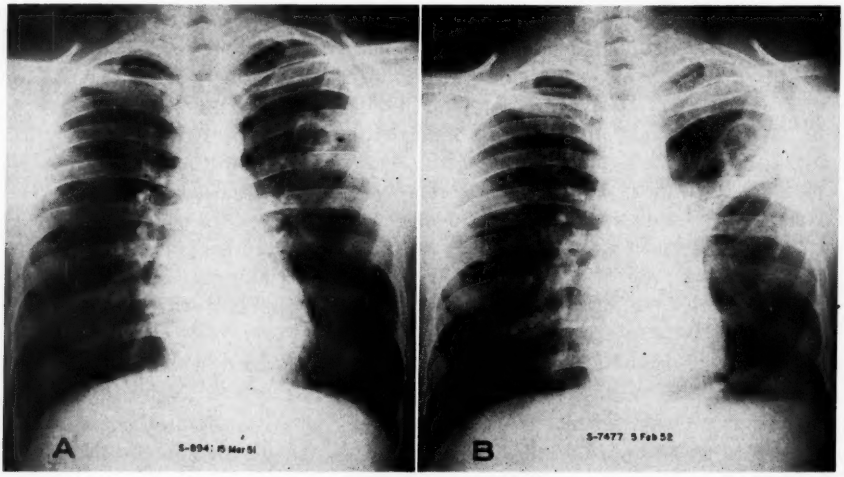

Fig. 1.—Case 1. *A*, Thin-walled coccidioidal cavity in left upper lobe. *B*, Large recurrent cavity after segmental resection.

CASE 2.—A 33-year-old white man stationed in Arizona was well until May, 1952, when he experienced a mild upper respiratory infection characterized by weakness, malaise, fever, and chills. Symptoms subsided except for the persistence of a chronic productive cough. Chest x-ray studies in December, 1952, revealed an infiltrate in the right middle lobe. By January, 1953, this area contained a 2.5 cm. cavity (Fig. 2). Sputum cultures grew *C. immitis*. The coccidioidin skin test was positive, and the complement fixation for coccidioidin was negative. In April, 1953, the right middle lobe and the anterior segment of the right upper lobe were resected. Examination of the specimen revealed a thick-walled cavity measuring 4.3 cm. in diameter. This cavity was close to the line of resection. Cultures from the cavity yielded *C. immitis*. Postoperatively the patient developed a bronchopleural fistula which required closed tube drainage. The empyema fluid contained hemolytic *Staphyloccoccus aureus* and *C. immitis*. After 3 months the fistula closed. The patient has continued to be asymptomatic.

CASE 3.—A 43-year-old white male physician was admitted to this hospital in 1949 with a 1-year history of chronic productive cough with hemoptysis. Six years previously

he had been stationed in the desert areas of Arizona and California and had an acute illness compatible with coccidioidomycosis. The coccidioidin skin test was strongly positive. Sputum cultures for *C. immitis* were negative. Coccidioidal complement fixation test was positive to a 1:18 titer. X-ray revealed a thin-walled cavity 5 cm. in diameter in the left lower lobe. In August, 1949, a left lower lobectomy was performed. A smear from the specimen revealed *C. immitis*. Postoperatively the patient developed a bronchopleural fistula and empyema. Subsequently he continued to have chronic chest pain and productive cough. Sputum cultures were negative for *C. immitis*. In December, 1950, the lingula of the left upper lobe was resected. In 1954, at another hospital, the left pneumonectomy was completed. After this resection there developed a bronchopleural fistula and an empyema infected with *C. immitis*. Sputum cultures were also positive for the organism. Recently, intravenous therapy with Amphotericin B has resulted in sputum conversion for the first time in several years.

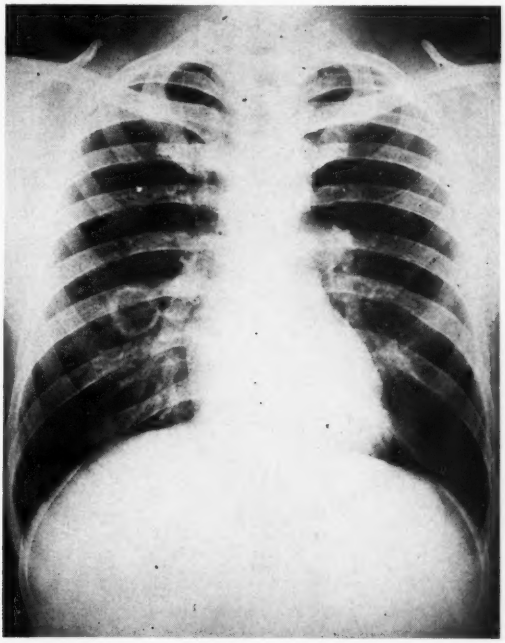

Fig. 2.—Case 2. Coccidioidal cavity in right middle lobe.

CASE 4.—This 24-year-old white woman suffered fatigability, weakness, and chest pain in 1951. X-ray examination revealed a "coin lesion" in the right upper lung. Coccidioidin skin tests were positive. The symptoms subsided, and the patient remained asymptomatic until February, 1956, when a chest roentgenogram revealed a 2 cm. thin-walled cavity in the right upper lobe. At this time the coccidioidin skin test was positive; the complement fixation tests were negative. In August, 1956, she had a wedge resection of the anterior segment of the right upper lobe. In October, 1956, closed tube thoracotomy was performed for pneumothorax secondary to bronchopleural fistula. After discharge, 1 month later, she noted chronic cough, episodes of hemoptysis, and pain in the right upper chest. She was re-admitted in April, 1957, with another cavity in the right upper lobe. The coccidioidin skin test continued to be positive. Sputum cultures were positive for *C. immitis*. Complement fixation tests for coccidioidin were positive 1:8. The patient was given a 6 weeks'

course of intravenous Amphotericin B with a total dosage of 2.2 grams. The productive sputum and chest pain disappeared and sputum cultures became negative. The cavity is unchanged. She was discharged in July, 1957, improved and has remained asymptomatic.

CASE 5.—In 1953, this 29-year-old white man had an acute respiratory infection of 2 weeks' duration following residence in the San Joaquin valley of California. One year later left chest pain and a productive cough developed. In February, 1955, x-ray studies revealed a cavity in the left upper lobe (Fig. 3, *A*). The sputum was positive for *C. immitis,* and the coccidioidin skin test was positive. In October, 1955, he experienced severe left chest pain, malaise, fever, and productive cough. It was noted that the cavity in the left upper lobe had enlarged (Fig. 3, *B*). Complement fixation test for Coccidioides was positive in a dilution of 1:4. Sputum culture grew *C. immitis.* In March, 1956, the anterior segment of the left upper lobe and the superior segment of the lingula were resected. The resected specimen contained a 5 cm. cavity.

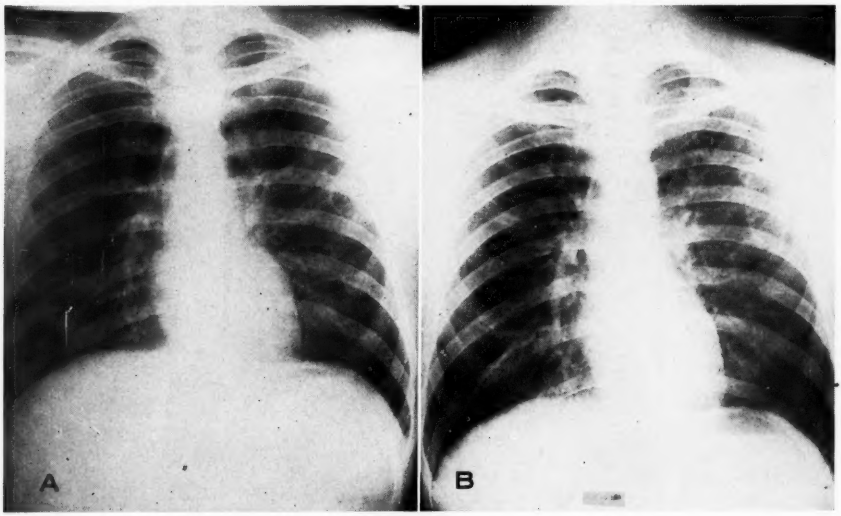

Fig. 3.—Case 5. *A,* Coccidioidal cavity in left upper lobe. June, 1955. *B,* Cavity enlarging. February, 1956.

The patient did well for 2 months after operation, when pain and productive cough recurred. X-ray examination revealed a recurrent cavity in the left upper lobe (Fig. 3, *C*). Sputum cultures remained positive for *C. immitis.* In March, 1957, left upper lobectomy and thoracoplasty were performed. Abnormal friability of the pulmonary artery made the operation extremely difficult. After operation, sputum cultures remained positive, and x-ray studies revealed a bronchopleural fistula with cavities in the left lower lobe (Fig. 3, *D*). Marked endobronchial disease was noted at bronchoscopy. The complement fixation test was positive in a dilution of 1:16, and the precipitin test was positive in a dilution of 1:8. The patient continued to have severe symptoms. In May, 1957, a 6-week course of Amphotericin B was administered intravenously, the dose totalling 2.7 Gm. After 1 week of therapy the sputum cultures became negative, and the symptoms subsided. Bronchoscopy revealed regression of the endobronchial disease. Two months after cessation of drug administration, the sputum cultures again became positive. A second course of Amphotericin B was initiated. The sputum cultures again became negative; and on Oct. 8, 1957, after 4 weeks of drug therapy, a left pneumonectomy was completed. Amphotericin was

continued for 2 weeks after operation until a total dose of 3.0 Gm. was administered. The resected specimen revealed multiple cavities containing viable organisms. Postoperatively the patient did well, and sputum cultures have remained negative. The patient has regained his weight, is asymptomatic, and gainfully employed.

DISCUSSION

Our indications for resection have generally agreed with those proposed by other authors.[4, 5] Surgery has been performed for relief of symptoms or for diagnostic reasons. The noncalcified solitary pulmonary nodule is a frequent indication for diagnostic resection, particularly in excluding a neoplasm. Asymptomatic cavitary lesions of unknown etiology are often resected for diagnostic

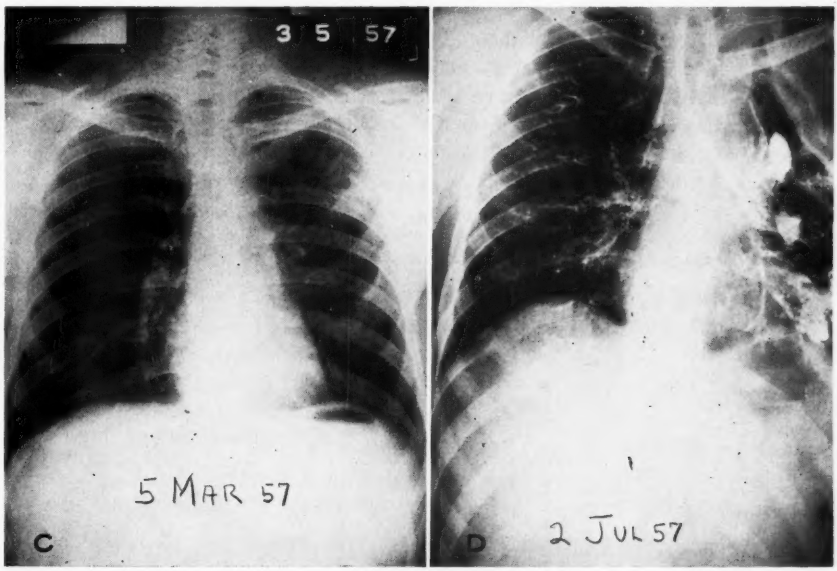

Fig. 3.—Cont'd. *C*, Recurrent cavity after segmental resection. March, 1957. *D*, Multiple cavitation after lobectomy. July, 1957.

reasons. These patients may come to surgery with the diagnosis of pulmonary tuberculosis. Enlarging cavities are removed to forestall the danger of cavity rupture, secondary infection, or hemorrhage. A number of patients present with moderate to severe symptoms as a result of their chronic infection. In some of these, there occurs local spread of the mycotic infection, as manifested by pulmonary infiltration or multiple cavitation. Other patients have symptoms from secondary infection or bleeding within the cavity or from rupture of the cavity into the pleural space. Patients having small asymptomatic cavities and sputum positive for *C. immitis* have been treated by close observation. If symptoms develop, resectional surgery is advised. The hazard of expectant treatment is not great in selected cases, and there are no reported instances wherein individuals with stable cavitary coccidioidomycosis have developed

dissemination.[3, 4] In addition, no human infection has occurred after contact with a patient having sputum containing *C. immitis.* This is due to the fact that the spherule, as found in the sputum, is not the infectious form of the organism even though the mycelial form may be present in the cavity wall.

It is undesirable to resect cavitary lesions in the presence of dissemination. Accordingly, we have utilized the serologic examination as an aid in eliminating this group. Smith[3] suggests that a complement fixation titer in excess of 1:16 should be regarded as suspicious of dissemination. One patient in this series had a complement fixation test of 1:80 prior to his initial operation. However, he had no other confirmatory evidence of dissemination. The presence of a strongly positive skin test in this patient was helpful in eliminating this possibility. Patients with coccidioidal empyema may have marked elevation of complement fixation titer without dissemination. With the appearance of empyema, the precipitin reaction may become positive. Surgery is not proposed for cavities observed to have been present for less than 6 months because of the remote chance of spontaneous closure. Cavities which have been present for longer than 6 months rarely close.

TABLE II. SURGICAL PROCEDURE AND COMPLICATIONS IN 100 CASES OF PULMONARY COCCIDIOIDOMYCOSIS

OPERATIVE PROCEDURE	TYPE OF LESION	COMPLICATION	
		RECURRENT PULMONARY CAVITY	BRONCHOPLEURAL FISTULA
	Cavitary		
Lobectomy	21	1	0
Segment and lobe	1	0	1
Segment	13	3	1
Wedge	13	1	0
	Noncavitary		
Lobectomy	1	0	0
Segment and lobe	0	0	0
Segment	7	0	0
Wedge	40	0	0
	Pyopneumothorax		
Wedge and decortication	3	0	1
Lobectomy and decortication	1	0	0

Experience with surgical treatment of cavitary pulmonary coccidioidomycosis indicates that postoperative complications occur more frequently than in other forms of chronic pulmonary suppuration (Table II). These complications have been related to persistent infection with the organism *C. immitis* as manifested by recurrent cavitation or bronchopleural fistula with empyema. Cavity recurrence has been reported by a number of authors.[6-10] These complications have occurred after resection of cavitary lesions. In this series of 48 cavitary resections, cavities recurred in 5 patients. In 2 additional cases, bronchopleural fistula with empyema followed the initial operation. Only one recurrence (5

per cent) followed lobectomy, whereas 22 per cent of the patients having segmental or wedge resection for cavity developed recurrent cavity or bronchopleural fistula. The propensity of these complications to follow procedures other than lobectomy seems to indicate that surgical dissection through diseased lung tissue has resulted in reactivation of the disease. Examination of the surgical specimens generally revealed satellite lesions in the vicinity of the cavity and granulomas in adjacent bronchi. Transection of such bronchi might result in bronchopleural fistula or an acute flare-up of the disease process in the bronchial stump with resultant seeding of adjacent lung tissue. Accordingly, at the Fitzsimons Army Hospital, lobectomy is the recommended surgical treatment for cavitary *C. immitis.*

Amphotericin B when administered in adequate dosage by the intravenous route appears to be a useful antifungicidal agent in man. Our use of this drug has been limited to the treatment of postoperative complications. In these instances sputum conversion has been obtained, and endobronchial disease has healed. When indicated, subsequent operation has been successful, although viable organisms were demonstrated within the resected specimen. To date this drug has not been used as an ancillary agent in the pre- and postoperative management of cavitary coccidioidomycosis. However, the routine administration of this or a comparable drug in the medical and surgical treatment of pulmonary coccidioidomycosis can easily be envisioned in the near future.

Follow-up information is available in 96 per cent of the patients. Two patients are dead. One died of coronary artery disease 9 years after resection. A second patient committed suicide 5 years after leaving the hospital. The remainder of the patients followed are living and well.

SUMMARY AND CONCLUSIONS

1. One hundred cases of patients with pulmonary coccidioidomycosis who were operated upon from 1947 to 1957 have been reviewed and analyzed.

2. Six of 27 patients (22 per cent) having segmental or wedge resection for cavitary coccidioidomycosis developed cavitary recurrence or bronchopleural fistula after operation. One of 21 patients (5 per cent) developed recurrent cavitation after lobectomy.

3. Lobectomy appears to be the method of choice in resection of cavitary pulmonary coccidioidomycosis.

4. Intravenous Amphotericin B is a fungicidal agent, effective in treatment of the complications of pulmonary resection for coccidioidomycosis.

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ANOMALOUS DRAINAGE OF THE PULMONARY VEINS INTO THE INFERIOR VENA CAVA

Report of an Operation

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As THIS is an uncommon anomaly of which there are only 35 cases reported in the literature with operation in only 3 instances, we have thought it would be of interest to report a case successfully treated by operation.

The anomalous drainage of the pulmonary veins can be total or partial. In the former, all the pulmonary veins empty in the right atrium, directly or through a systemic vein. In the latter, some veins end normally at the left atrium and others go directly or indirectly to the right atrium. The most frequent partial anomalies occur on the right side and generally are associated with an interatrial septal defect. In order of frequency, the site of anomalous communication may take place at the superior vena cava, right atrium, left innominate vein, and, less frequently, at the portal vein and inferior vena cava. The frequency with which these anomalies are reported is increasing since Brody[1] (1942) collected 106 cases in the literature. In 1949 they increased to 133. Of this total only 4 were communications of the pulmonary veins with the inferior vena cava, and all 4 were necropsy findings. Dotter, Hardisty, and Steinberg[2] report the first 2 cases diagnosed in life.

From a physiopathological point of view this developmental anomaly is a left-to-right shunt in which the oxygenated blood drains into the inferior vena cava, continues to the right atrium, right ventricle, and recirculates in the lung. When it is a single anomaly, there are no or few symptoms, depending on the volume of blood that recirculates through the lesser circuit. According to Brody, 50 per cent of the blood should arrive at the right heart for symptoms to become apparent. In general this does not occur because frequently there is a hypoplasia of the diseased side with hyperplasia of the normal side.

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Of 9 cases studied by Steinberg, 6 were asymptomatic and the diagnosis was made radiologically. It is this author's opinion that the presence of dyspnea or bronchopulmonary alterations should make one suspect the presence of communications of the systemic circulation with the lung. These suggestive symptoms are dyspnea and sensation of fatigue. The objective signs are variable. Sometimes there is a systolic murmur at the pulmonary focus. The electrocardiogram can be normal or may indicate complete or incomplete bundle branch block, or right ventricular strain. In 8 cases of Steinberg's series it was normal. The x-ray findings show different grades of increase of the right chambers, pulmonary artery, and hilar branches. The right atrium is prominent and, frequently, the hypertrophy of the left lung displaces it toward the right hemithorax. Dotter and his colleagues[2] described the presence of a shadow with vascular density going from the pulmonary hilum to the cardiophrenic angle, parallel to the right auricle. The left lung appears well vascularized, even plethoric, and generally larger than the right lung that can even be hypoplastic.[3]

The angiocardiographic study is informative but not indispensable for diagnosis. It visualizes the pulmonary vein in its anomalous communication with the inferior vena cava and the reopacification of the right atrium and ventricle with the pulmonary artery, indicating the existence of a left-to-right shunt. Tomograms are of importance because they reveal the pulmonary vein and its branches in its anomalous direction. Heart catheterization demonstrates a higher oxygen saturation in the inferior vena cava when compared with the superior vena cava and right atrium.

Steinberg states that the asymptomatic cases do not require surgical correction; but we should not forget that these patients are in the same situation as if they had had a pneumonectomy, and are consequently exposed to a fatal end with any diffuse pulmonary or pleural disease. We believe that the surgical correction involves little risk and should be done to restore a lung to active function.

The number of patients operated upon is extremely low. In an extensive review of the literature we have found the report of only 3 cases corrected surgically. We shall refer to them briefly. Kirklin,[4] based on the experimental work of Gerbode and Hultgren,[5] advocates the disconnection of the anomalous vein at its entry into the vena cava and its reimplantation on the left auricle. He reports on 6 cases, one of which was the anomaly under study associated with an interatrial septal defect in which he made a reimplantation with atrioseptopexy after Bailey.[9] Risch and Hahn[7] from Leyden, Holland, reported 25 patients with an anomalous connection of the pulmonary vein who were operated upon. In 1 of their cases the drainage occurred at the inferior vena cava, 2 cm. from the right atrium, and was associated with an interatrial communication. A reimplantation was done and the septal defect was repaired with open-heart technique under hypothermia. Ehrenhaft and co-workers[6] reported a similar case.

CASE REPORT

Our case report follows.

A. L. V. (581586), a 41-year-old woman, complained of dyspnea and cardiac palpitations. The past history was of no significance. The only prominent physical finding was a systolic murmur at the second and third intercostal space on the left that was best heard in the recumbent position; on standing it practically disappeared. The electrocardiogram revealed a normal electrical axis with an incomplete bundle branch block. The chest films and tomograms revealed a shadow of diffuse borders placed on the right side of the heart, between the hilar region and the costodiaphragmatic sinus. The heart was normal in size. The conus of the pulmonary artery was enlarged. The

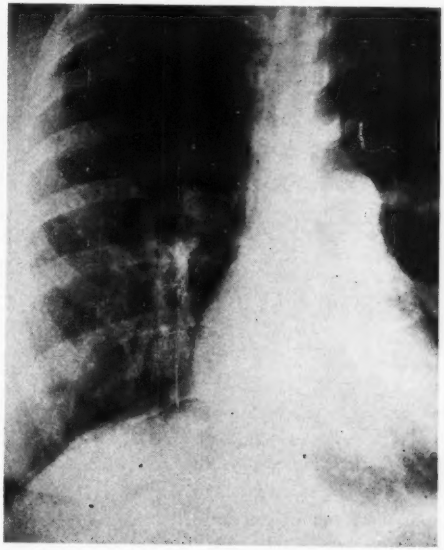

Fig. 1.—Chest roentgenogram which demonstrates a shadow of diffuse borders placed on the right side of the heart between the hilar region and the costodiaphragmatic sinus.

radiologist considered the possibility of an arteriovenous pulmonary fistula. This was not confirmed in the tomograms. The patient was operated upon July 10, 1958, with the diagnosis of a right pulmonary arteriovenous fistula.

Operative Findings.—From the inferior border of the right lung, there was a vascular bundle, directed to the posterior region of the diaphragm, composed of a vein of 1 cm. in diameter with two arterioles originating in the superior diaphragmatic arteries and ending in the lung parenchyma. The vein connected with the inferior vena cava. The bronchial and arterial anatomy at the hilum was normal; no pulmonary vein was found going to the heart and this was confirmed by opening the pericardium. The left pulmonary veins were normal. The lung was divided into two lobes; the superior lobe representing four fifths of the lung, and the inferior lobe in a posteromedial position was one fifth.

Operation.—Both arterioles were divided. The pulmonary vein was dissected and divided at the junction with the inferior vena cava. Previously the pulmonary artery had been compressed. The distal end of the vein was ligated. The proximal end was passed

through a hole in the pericardium and sutured to the left atrium. The wall of the left atrium was grasped with a curved Potts clamp and an opening of 1 cm. was made. Sutures were made with 5-0 non-traumatic silk. No leak was observed when the clamp was removed.

The postoperative course was uneventful. Tomograms taken 4 months later showed a vascular shadow at 7.5 and 8 cm. going to the mid-portion of the heart corresponding to the transplanted vein. Bronchographic studies confirmed an anomaly in bronchial distribution, only two lobes existing. Biopsy of both arterioles confirmed the microscopic finding.

Fig. 2.

Fig. 3.

Fig. 2.—Preoperative tomogram of the case.

Fig. 3.—Postoperative tomogram that shows a vascular shadow at 7.5 and 8 cm. going to the mid-portion of the heart corresponding to the transplanted vein.

SUMMARY

In our case we can say in summary that besides the anomalous connection of the pulmonary vein to the inferior vena cava, there were two systemic arteries entering the lung at the right inferior lobe branching off from the superior diaphragmatic arteries and an anomaly in bronchial distribution. This case resembles that reported by Park[10] (1912).

The postoperative course of the patient to date has been uneventful and satisfactory.

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A LIGHT WEIGHT NEEDLE HOLDING FORCEPS

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IN the course of repairing numerous femoral arteriotomies after bypass perfusion, no needle holder has proved entirely satisfactory. Those with good grips are too heavy, while the light instruments allow the needle to swivel.

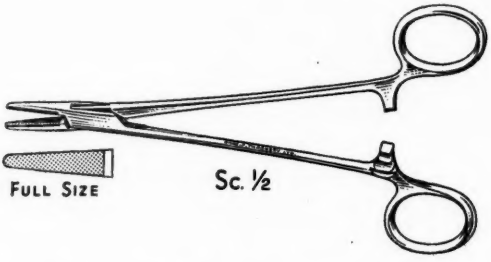

Fig. 1.—Light weight needle holding forceps.

The needle holder shown in the illustration overcomes these disadvantages. The over-all length is 6 inches, and the instrument weighs exactly 1 ounce; the joint is of the box type, and the jaws are fitted with Tungsten carbide inserts, which effectively prevents any swivelling of the needle during suturing.

Although this needle holder was designed primarily for the purpose mentioned above, it is equally valuable in any relatively superficial vascular work, as well as in suturing in very small infants.*

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*The instrument was made for us and can be supplied by Chas. F. Thackray, Ltd., Leeds, England.

Announcement

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Notice of Annual Meeting

The Forty-second Annual Meeting of The American Association for Thoracic Surgery will be held April 16, 17, and 18, 1962, in St Louis, Missouri. Headquarters will be The Chase-Park Plaza Hotels.

Requests for Hotel Reservations

These should be mailed directly to Mr. T. J. Robinson, Executive Assistant Manager, The Chase-Park Plaza Hotels, St. Louis 8, Missouri. Please mention this Association, the type of accommodation desired, the date, and approximate hour of arrival and departure. If accommodations are desired elsewhere please communicate directly with the hotel of your choice.

Material for the Program

SCIENTIFIC PAPERS.—Abstracts of all papers proposed for presentation at the 1962 meeting *must be received on or before* **November 15, 1961,** otherwise they will not be considered by the Program Committee. These should contain *not more than* **200** *words* that accurately and clearly reflect the content of the completed paper. They should be labeled "For Thoracic Surgery Forum" or "For Regular Program," depending upon the nature of the subject matter. The Forum is primarily for the presentation of short papers on current thoracic research, experimental problems, anatomical studies, and modified or new surgical techniques.

Six, repeat, six clearly legible copies of each abstract must be sent to Miss Ada Hanvey, Administrative Assistant, The American Association for Thoracic Surgery, Suite 311 Carondelet West, 7730 Carondelet Avenue, St. Louis 5, Missouri.

Essayists selected for the program are reminded that papers presented at the meeting are to be published in the Journal and *must* be handed to the secretary *immediately* after their presentation. The maximum length allowed for Forum papers is 3,000 words without illustration. If illustrated, an appropriate number of words must be subtracted.

Applications for Membership

Applications for Associate Membership in the Association must be received by the Membership Committee *not later than* **November 15, 1961,** otherwise the application will be deferred for consideration until the 1963 meeting.

Applicants must be sponsored by three Active or Senior Members of the Association. In addition to the signature on the application form, the sponsors will forward a *separate* letter concerning the applicant directly to:

Charles K. Kirby, M.D.
Chairman of the Membership Committee
3400 Spruce Street
Philadelphia 4, Pennsylvania

Sponsors are reminded that *new* letters of recommendation must be forwarded to the Chairman of the Membership Committee should their applications be held over for reconsideration a *second year.*